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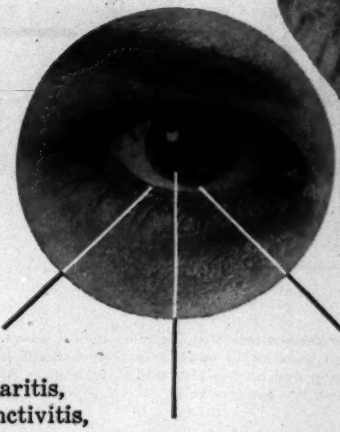

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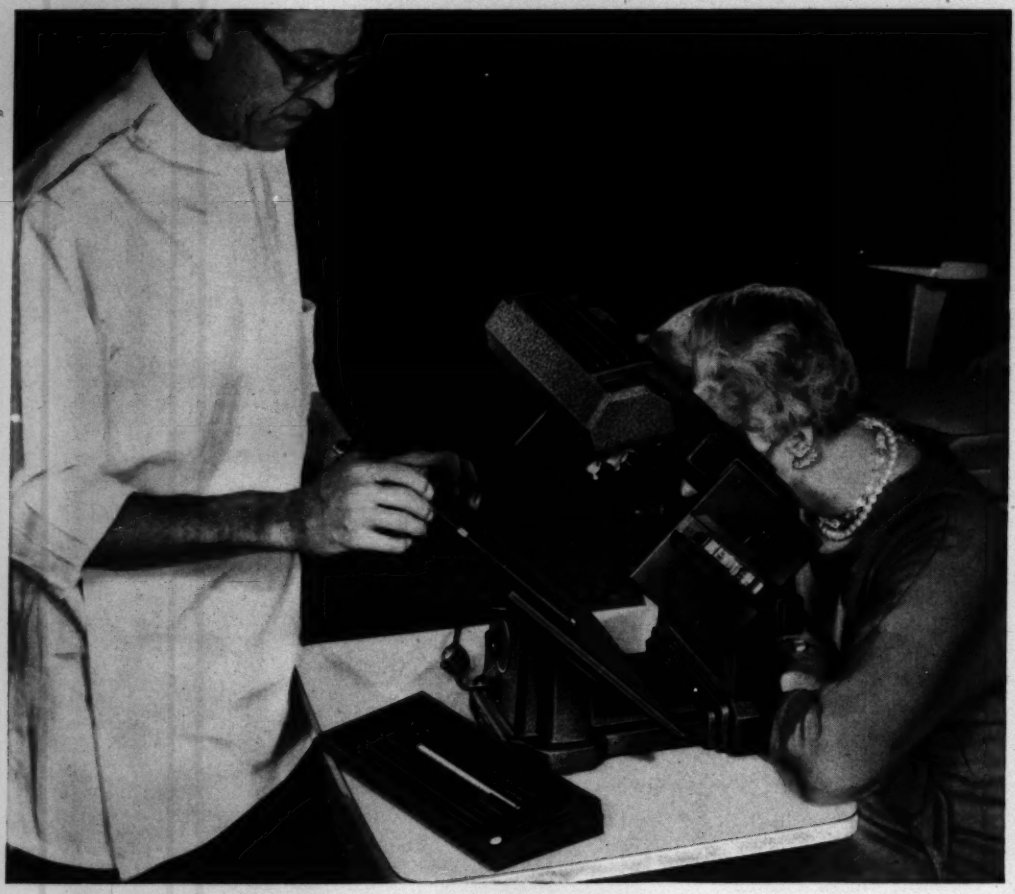

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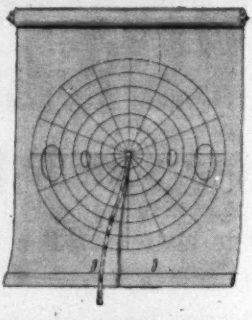

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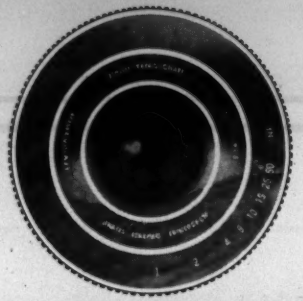

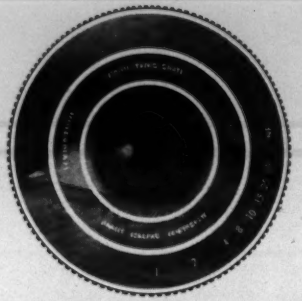

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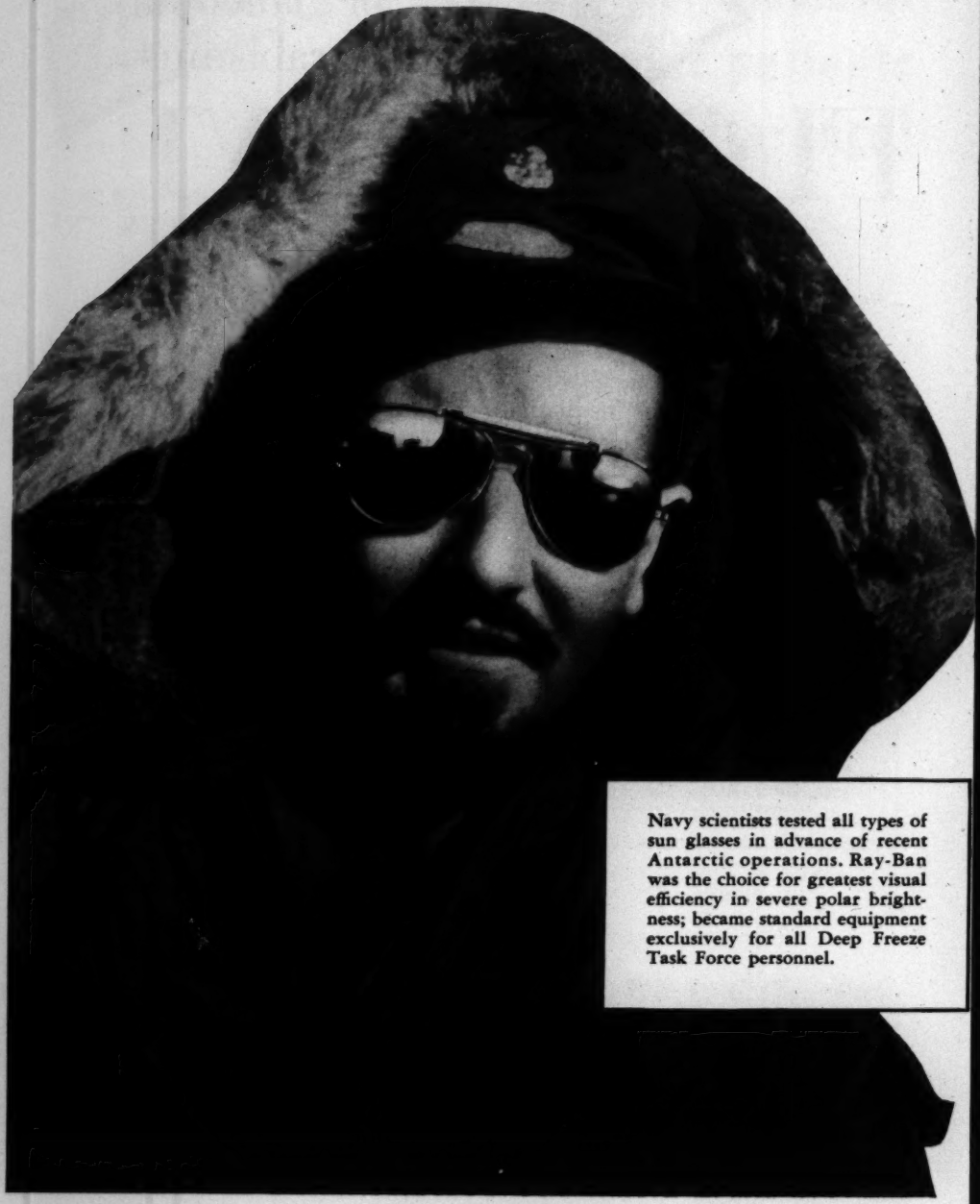

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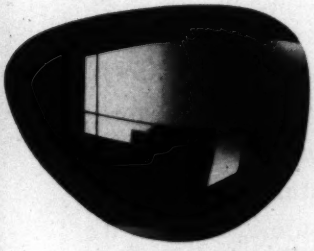

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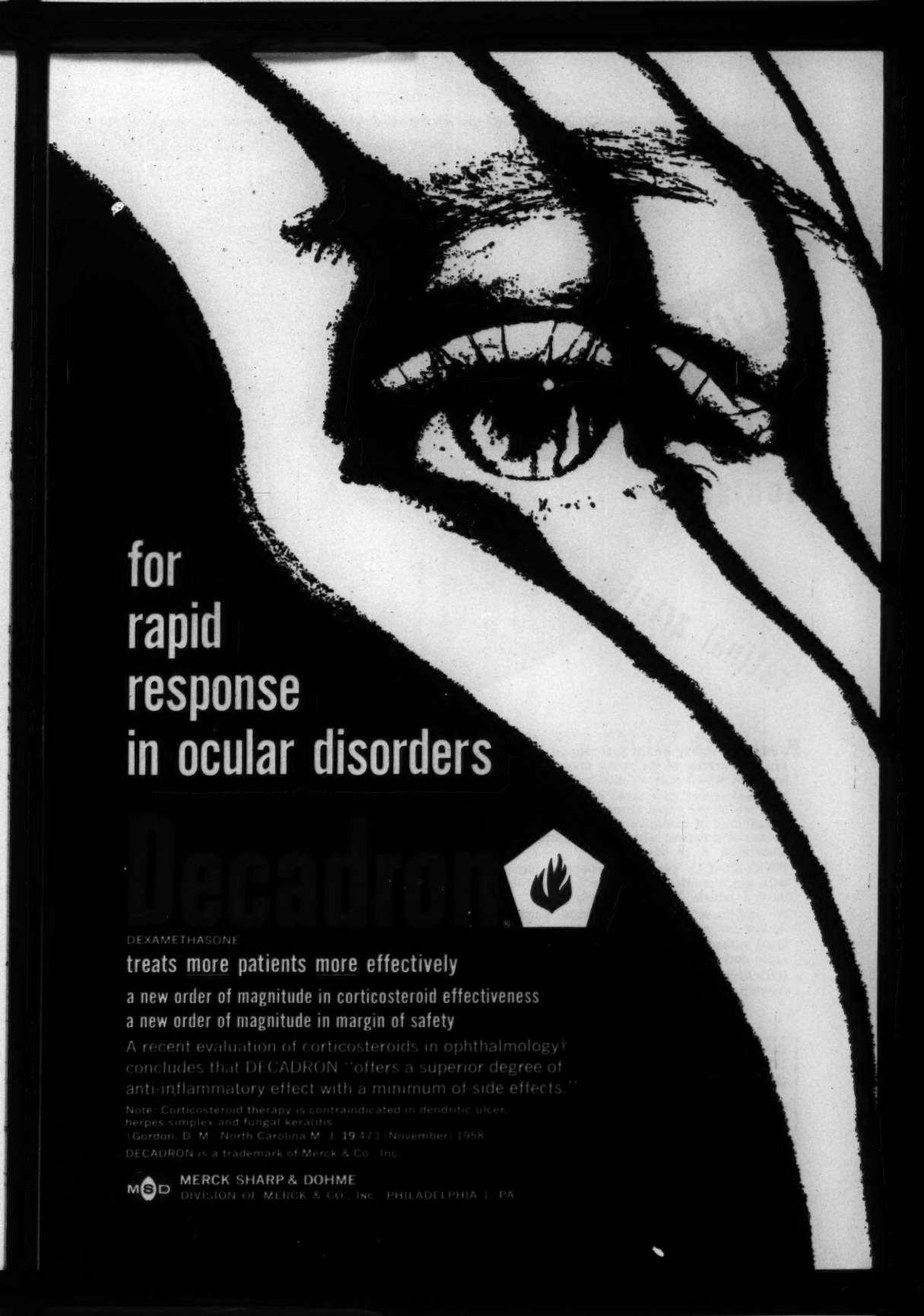
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1. *Cecil's Textbook of Medicine*, 7th ed., 1947, p. 1287.
2. *Ibid.*, p. 1598.
3. *Am. J. Ophth.* 42:771, 1956.
4. *Am. J. Digest. Dis.* 22:5, 1955.
5. *Med. Times* 84:741, 1956.

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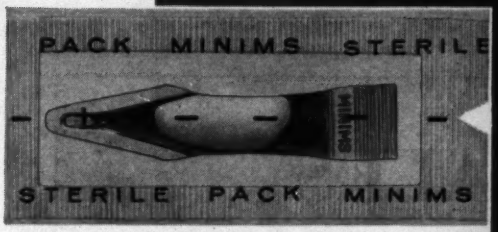

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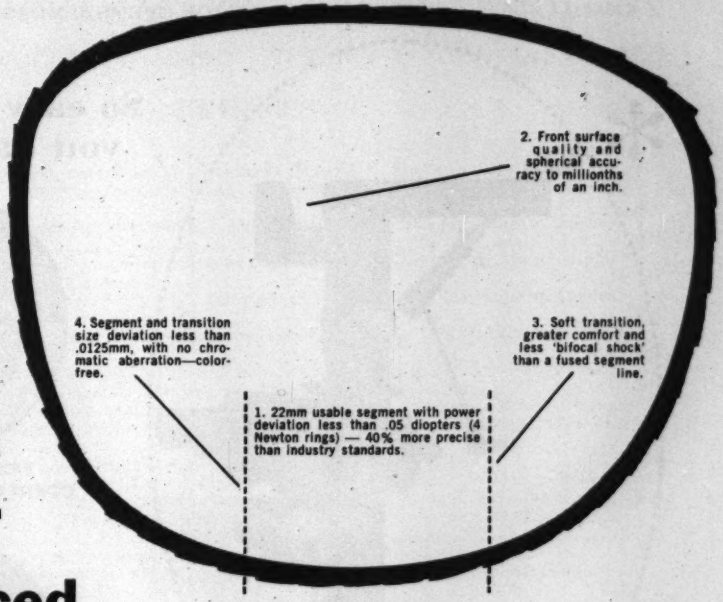

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AMERICAN JOURNAL OF OPHTHALMOLOGY

VOLUME 47 APRIL, 1959 NUMBER 4

OPHTHALMODYNAMOMETRY AND CAROTID ARTERY THROMBOSIS*

HAROLD F. SPALTER, M.D.
New York

The appearance of a spontaneous arterial pulse was first noted by Donders[1] in 1855, shortly after the introduction of the ophthalmoscope. Thereafter, clinical observations revealed that in glaucoma where the intraocular pressure is high, a spontaneous pulsation of the central retinal artery was frequently seen. It was also noted that pressure upon the globe with the fingers elicited an expanding and collapsing pulse. From this came the concept of converting the eye into its own blood-pressure apparatus with the diastolic and systolic end-points represented by the onset and collapse of pulsation of the central retinal artery.

In 1917, Bailliart[2,3] devised a simple standardized tension spring instrument called the ophthalmodynamometer for applying and recording increasing pressure upon the eye. The pressures are recorded in grams of water and Bailliart prepared a conversion table to millimeters of mercury based on initial ocular tension. The technique of ophthalmodynamometry gained many enthusiasts in the 1920's and 1930's. A large number of reports appeared in the literature here and abroad. They attempted to prove absolute values for retinal artery pressure and absolute ratios between systemic blood pressure and retinal artery blood pressure. The investigators, however, failed to agree on any definite values. Priestly Smith[4] showed the fallacy of comparing measurements between individuals, as the pressure would vary considerably according to the size of the globes and their scleral rigidity. Duke-Elder[5] postulated that what is actually being measured is not the central retinal artery pressure but the pressure at a point somewhere between the ophthalmic artery and the central retinal artery. Koch,[6] in an extensive review of the literature in 1945, stated that no definite conclusions could be reached about the value of ophthalmodynamometry from the reports in the literature.

The method was thus neglected and essentially discredited as a reliable tool for comparing pressures between individuals, for estimating systemic blood pressure, or for providing absolute values in statistical studies. Even at this time, however, it was recognized that the technique might prove to be of value in comparative measurements between the two eyes of the same individual; for then the factors of variability between eyes could be minimized and a fairly adequate comparison made between the units of extraocular pressure required to achieve the same end-points of onset and collapse of pulsation in the two eyes of the same individual.

With this approach in mind, the technique to be clinically useful must provide answers to the following questions: Do differences in measurements exist between the two eyes in normal individuals? If differences exist what is their range of normal variation? Are the differences reproducible? Do differences exist in disease states that exceed the range of normal variation?

* From the Institute of Ophthalmology, Columbia Presbyterian Medical Center. I wish to thank Dr. Elliot Weitzman and the staff of the Neurological Institute Columbia Presbyterian Medical Center, for their help in obtaining patients for presentation.

Present study

Fifty normal patients were studied at the Eye Clinic at Presbyterian Hospital. They were selected at random from a group of patients desiring refraction. All of the patients had normal intraocular pressure. The method of ophthalmodynamometry has been well described and a brief review will suffice.

The instrument used is the Bailliart ophthalmodynamometer of which several models are available. The model with the dial face and automatic stop is probably the most compact and easiest to manipulate.

The patient is sitting or supine (fig. 1). Mydriasis is helpful but not necessary if the patient is co-operative and a clear view of the central retinal artery can be obtained. Topical anesthesia with pontocaine drops is indicated in most instances, although many patients can easily tolerate the procedure without anesthesia.

The convex foot-plate is applied over the lateral rectus insertion on the sclera about one centimeter from the limbus. The instrument is held and maintained in the horizontal position to eliminate the effects of gravity. Pressure is then gradually applied while observing the central retinal artery or one of its major divisions on the disc.

The onset of arterial pulsation is quite abrupt and simulates the appearance of a flashing red streak as the red color of the vessels appears and disappears in collapsible pulsation at the same rate as the heart beat.

Fig. 1 (Spalter). Ophthalmodynamometer (Bailliart) in position over lateral rectus. Single examiner holds instrument and ophthalmoscope.

At this point the pressure (diastolic) is noted on the instrument. Pressure is then continued until the pulsation ceases and the artery is collapsed (systolic pressure) and the pressure reading again noted.

The technique is simple and requires less time than taking the systemic blood pressure. It is easily mastered after a small amount of practice. I have found it easier to observe the fundus and apply the instrument myself, although some workers prefer using two examiners. The end-points are usually easily reproducible and all measurements are repeated three to five times to provide a check on their accuracy.

One difficulty encountered is in recording the systolic end-point. When the complete collapse of pulsation occurs, continued pressure on the globe permits the pulse to reappear within one or two seconds. Apparently this is due to a fall in intraocular pressure as fluid is driven out of the eye by the pressure of the instrument, thus the total pressure against the artery falls below the systolic pressure, permitting the pulse to reappear. Therefore, if the systolic end-point is missed it is best to wait 10 minutes for the intraocular equilibrium to be re-established before attempting another measurement. Diastolic measurements may be repeated without this interval of waiting.

Other points of caution to observe include misinterpretation of the venous for the arterial pulse, not a great problem for the experienced ophthalmoscopist. Cardiac irregularities may prevent recognition of distinct end-points. Poor visualization of the artery because of opacites in the cornea or media invalidates the technique.

The contraindications to the technique are relatively few and self-evident. These include retinal disease or high myopia where increased extraocular pressure might induce retinal detachment, recent ocular surgery, central retinal artery or vein thrombosis, or glaucoma. No reports exist in the literature indicating any serious complications following the use of the ophthalmodynamometer.

Age Range: 14-75

Intraocular tension: normal in all subjects

Maximum Difference in Readings Between Two Eyes

Systolic: 15 mm.
Diastolic: 8 mm.

Average Difference In Readings Between Two Eyes

Systolic: 5mm.
Diastolic: 3mm.

Maximum % Difference Between Two Eyes

Systolic: 15%
Diastolic:10%

Average % Difference Between Two Eyes

Systolic: 5%
Diastolic:4%

Fig. 2 (Spalter). Ophthalmodynamometer measurements in 50 normal subjects.

The only direct complication of the technique is a subconjunctival hemorrhage at the site of the application of the foot-plate, but this is rare and I have never encountered it in over 200 eyes examined.

The summary of the findings in 50 normal subjects measured by the technique just outlined is found in Figure 2. These results compare favorably with those found in other studies.[7-9] All measurements were repeated several times and were reproducible within 5.0 mm. Pressures in any one individual will vary according to the systemic blood pressure at the time of measurement. However, the percentage differences remain essentially unchanged. Spot checks were made by another observer on many of the controls and the values were essentially equal to my own measurements.

The percentage difference for either the systolic or diastolic pressure is calculated by the simple formula of

$$\frac{\text{higher RAP} - \text{lower RAP.}}{\text{higher RAP}}$$

Some authors[8] record only the diastolic pressure; however, clinical situations have been reported where only the systolic measurements reveal a significant difference, and therefore I feel that whenever possible both systolic and diastolic determinations should be made.

An apparently reliable range of normal variation having been established, what should be considered a significant difference? Since the upper limit of variation in this study is 15 percent in the normal subjects, a minimum difference of 25 percent or greater in systolic or diastolic levels between the two eyes would be adequate to denote a "significant" difference; that is, a difference exceeding the range of normal variations. Other authors[8,10] have used lower levels, for example, 10 mm. or a 10 percent systolic and 15 percent diastolic difference. It is worth while to note that with diastolic pressures below 30 mm., a correspondingly larger pressure difference should be used as the minimum for a significant difference.

Differences in disease states

It would be anticipated that in disease states in which ocular symptoms are present, possibly reflecting a decreased blood flow and blood pressure to one eye, ophthalmodynamometry might prove to be of value. In carotid artery thrombosis, temporal arteritis, Takayasu's (pulseless) disease, and arteriosclerotic optic atrophy, visual symptoms, often unilateral, occur frequently enough to bring the patient to the ophthalmologist, often as the first physician consulted. In each disease decreased blood flow to the optic nerve or retina is the major cause of the visual disturbance. Such a group of diseases then provides a fertile field of study for the clinical application of ophthalmodynamometry. Since no cases of temporal arteritis, pulseless disease, or arteriosclerotic optic atrophy have been encountered during the preparation of this study, the report will deal only with cases of carotid thrombosis.

Ophthalmodynamometry in carotid artery thrombosis

Carotid artery thrombosis is a relatively "new "disease in that the awareness of its true frequency has developed only over the past five to 10 years. Prior to the advent of cerebral arteriography there were only scat-

tered reports of spontaneous thrombosis of the carotid artery and the diagnosis was rarely made before death.

Fisher,[11a, 11b] in 1951, called dramatic attention to the incidence of carotid artery disease by finding that in a consecutive series of 432 autopsy cases, in which there was cerebral disease and in which the brains were removed and the carotid arteries studied, 6.5 percent had complete occlusion of one or both carotids and 3.0 percent had severe stenosis of the carotids. Amazingly enough this was approximately the same incidence as cerebral hemorrhage and hypertensive arteriosclerotic encephalomalacia in the same series and only a slightly less incidence than cerebrovascular thrombosis or so called cerebral vascular accidents or strokes. It thus appeared that many diagnoses of cerebrovascular accidents ante mortem were, in actuality, examples of unrecognized carotid artery disease. Since this report, increasing numbers of pathologic studies have appeared in the neurologic literature as the carotids are more carefully studied post mortem, and the diagnosis is being more actively considered clinically in the differential diagnosis of cerebral disease.

The etiology in the majority of cases is atherosclerosis. Rarely the carotid is congenitally stenosed or absent,[12] and a few cases of nonspecific arteritis, as well as tumors infiltrating and occluding the artery, have been reported. The age range is from the fourth to the seventh decade with a peak incidence in the fifth, slightly earlier than the peak incidence of other cerebrovascular disease. Spontaneous thrombosis has been reported in the first, second, and third decades but this is rare.[13]

The site of occlusion in about 80 percent of the cases reported is just at or beyond the bifurcation of the common carotid artery in the neck, with the remainder of the cases having the occlusion initially in the intracerebral portion of the carotid usually at the siphon.[14]

The symptomatology of the disease can probably best be understood in terms of its pathogenesis (fig. 3). Two things of consequence may happen to the diseased carotid artery. One is a slow progressive occlusion of the lumen or, secondly, an acute thrombosis. Clinically the latter is manifested by an onset with an acute episode simulating a stroke and the former, on the other end of

I. ACUTE (Incidence: 35%)
- Sudden onset without warning
- Identical to severe stroke
- Loss of consciousness, hemiplegia, aphasia
- Mortality 35%
- May simultaneously thrombose ophthalmic artery

II. TRANSIENT EPISODES (Incidence: 40%)
- Lasting several minutes to several hours
- Frequency variable
- Symptom complex often reproducible
- Intermittent mono or hemiplegia, aphasia, amaurosis, sensory loss
- No neurologic or ophthalmologic deficit between episodes
- May cease spontaneously or develop permanent damage

III. SLOWLY PROGRESSIVE COURSE (Incidence: 25%)
- Simulates brain tumor
- Increasing paresis, psychic disturbance, visual field loss
- May be interrupted by development of collaterals

Fig. 3 (Spalter). Natural history of carotid artery disease. Thrombosis or insufficiency.

the clinical spectrum, presents with progressive cerebral involvement mimicking the picture of a brain tumor. Occupying the largest part of the clinical spectrum is the syndrome of intermittent carotid artery insufficiency with recurrent transient episodes of neurologic and/or visual disturbance.

The acute attacks occur in about 35 percent of the cases and present suddenly without warning. The clinical picture is essentially indistinguishable from that of a cerebrovascular accident with hemiparesis, aphasia, loss of consciousness, homonymous field defects. About one third of this group dies as a consequence of the initial insult. These cases may rarely be associated with simultaneous loss of vision in the eye on the same side as the thrombosis. This finding helps to implicate the carotid artery as the site of the pathologic alteration and is due to an extension of the thrombus into the ophthalmic artery.

The second group consisting of about 40 percent of the cases is characterized by intermittent episodes of hemiparesis or monoparesis, transient sensory defects (cerebral and peripheral), and intermittent amaurosis on the ipsilateral side (in about 20 to 30 percent of cases). The episodes may occur several times a day to several times a week, month, or year. They may last from a few minutes to several hours, with the visual loss rarely lasting more than five minutes. Between attacks of carotid insufficiency the patients are asymptomatic and usually have no demonstrable neurologic or ophthalmologic abnormalities. The attacks may cease spontaneously if adequate collaterals develop to circumvent the arterial block, or the patient may develop permanent cerebral or visual damage.

The third group has a progressive course closely mimicking the clinical picture of an expanding intracranial lesion. This form of carotid disease occurs in approximately 25 percent of cases and presents a particularly difficult diagnostic problem to the clinician.

The symptoms of carotid artery disease in their approximate order of frequency are shown in Figure 4. The symptoms may present in any combination and in an acute, intermittent, or slowly progressive course as discussed previously.

In a disease with such protean manifestations and a varying course and which can easily mimic other diseases in the neurologic and ophthalmologic spheres, how is the diagnosis made? For here the important consideration is that, with an early diagnosis, present surgical and anticoagulant techniques

1. Hemiplegia or Hemiparesis
2. Aphasia
3. Psychic Disturbances - premature senility; psychoneurosis; memory loss
4. Headache - most often generalized
5. Sensory Disturbance - hemianesthesia; hemihypalgesia; paresthesias of one extremity
6. Visual Disturbance- intermittent amaurosis; ipsilateral blindness; homonymous field loss; optic atrophy;
7. Convulsions - focal or diffuse
8. Coma
9. Monoplegia or Monoparesis

Fig. 4 (Spalter). Symptoms of carotid artery disease.

hold the possibility of significant improvement and alteration of the normally devastating course of the disease. Recent reports[15-17] indicate that reconstructive surgery on the thrombosed carotid by direct surgical approach to the carotid in the neck with thrombectomy, endarterectomy, and arterial grafting may prove to be of great success, particularly in cases with an early diagnosis.

The history and physical findings are not of much value except where amasesis reveals intermittent unilateral visual symptoms (especially amaurosis fugax) associated with neurologic disturbances, the single most characteristic historical finding in carotid disease. Arteriography plays a dominant though limited role in the diagnosis. The first series of diagnoses of carotid thrombosis ante mortem was based on the initial arteriographic investigations of Moniz.[18] Today, we recognize that the use of the carotid arteriogram is severely handicapped by the hazards of dye injection into the intracerebral circulation, especially in the older age group. In particular, in suspected carotid artery disease, additional manipulation of the artery may induce additional thrombus formation and completely occlude an already severely stenosed vessel. Numerous cases of increasing paresis, shock, and even homolateral blindness have been reported following cerebral arteriography.[19] The risk of the technique is often considered too great and therefore its usefulness is limited.

Palpation of the carotid artery either through the skin of the neck or by the oropharyngeal approach is not considered reliable by most observers.[20] The presence of a pulse does not rule out carotid disease, and its absence is insufficient evidence for definitive diagnosis.

Recently investigation of carotid disease by means of the electroencephalogram and the effects of postural change on the recordings has been carried out.[21] The success of the technique has varied according to different reports and more work is being done in this area.

A simple, reliable, and hazardless method of diagnosis may be ophthalmodynamometry. The physiologic justification for the use of the ophthalmodynamometer in the diagnosis of this vascular disorder may be found in Sweet's experiments.[22] In 1948, Sweet demonstrated that surgical occlusion of the common carotid artery resulted in a large pressure drop in the internal carotid artery as shown by direct intra-arterial measurements in the neck. In subsequent studies[23] it was shown that a fall of 60 to 90 percent in internal carotid artery pressure was transmitted to the intracranial internal carotid even to its branches with a diameter of 0.5 mm.

It would be expected then that the pressure in the ophthalmic artery, as the first major branch of the intracerebral internal carotid, would show a similar pressure drop. It has not been possible actually to cannulate the ophthalmic artery in man to prove this. However, this assumption is supported by the work of others.

In 1956, Svien and Hollenhorst[8] studied 11 patients in the immediate postoperative period following surgical ligation of the intracervical carotid artery and showed a difference of between 22 to 71 percent in retinal artery pressure readings between the occluded and nonoccluded sides. Other observers[24] have shown that merely pressure upon the neck will result in a significant fall in retinal artery pressure on the same side. Therefore, measurements by ophthalmodynamometry becomes a sensitive index of obstruction to flow in the carotid artery.

Thus, in spontaneous thrombosis of the carotid artery, a significant fall in ipsilateral retinal artery pressure would be anticipated, reflected in a significant difference between the opthalmodynamometer readings on the occluded and nonoccluded sides. Indeed this has been found to be the case. In the first report Miletti,[25] in 1946, reported one case with positive dynamometric readings. Thomas and Petrohelos,[9] in 1953 reported on eight cases. Four more cases were reported in 1956 by Svien and Hollenhorst[8]; seven cases by

PATIENT	AGE	SITE	TIME OF OBSERVATION	PATENT	THROMBOSED	%DIFFERENCE
A.J.	58	bifurcation on left	48 hours	130/70	60/30	54/57
A.K.	69	bifurcation on right	1 week	/80	/40	50
W.G.	49	1 cm. above bifurcation on right	2 weeks	/70	/25	64
D.M.	66	bifurcation on right	2 weeks	105/60	58/28	45/53
J.K.	39	1 cm. above bifurcation; right	3 weeks	125/70	90/48	28/31
M.D.	66	bifurcation on left	4 weeks	/65	/10	85
E.B.	65	2 cm. above bifurcation;right	4 months	105/60	30/20	71/66
M.D.	57	1 cm. above bifurcation;left	5 months	120/60	50/30	58/50
J.L.	50	bifurcation on left	2½ years	120/82	75/30	38/52
R.R.	60	1 cm. above bifurcation;left	3 years	125/62	125/60	0/3
A.H.	45	beyond ophthalmic artery on right	1 week	70/30	110/55	36/45
H.H.	50	beyond ophthalmic artery on left	3 years	110/42	120/48	8/12

Fig. 5 (Spalter). Ophthalmodynamometry findings in 12 cases of carotid artery thrombosis proved by arteriography.

Heyman, Karp, and Bloor[26]; and five cases by Wood and Toole[10] in 1957. Most recently three cases have been reported by an Allen,[17] et al., in 1958. Additional case reports may be found in the German literature.[27]

From the ward and private patients at Presbyterian Hospital Neurological Institute and by the co-operation of discharged patients who agreed to return to be studied, I was able to obtain 12 cases* of spontaneous carotid artery thrombosis for ophthalmodynamometric measurements. All the cases were documented as carotid thrombosis either by arteriography or by surgical exploration. None of them could be shown to have any demonstrable cause for the thrombosis other than presumed atherosclerosis. The patients ranged in age from 39 to 69 years, with three females and nine males. A summary of the pertinent findings is presented in Figure 5. The percentage difference recorded is the smallest difference found, either systolic or diastolic.

In 10 cases in which the thrombosis was found proximal to the ophthalmic artery only one patient, R. R., failed to show a significant difference. This patient was measured three years after the occlusive episode and his disease had not progressed. We can assume that, in this period of time, adequate collateral circulation had developed to eliminate any pressure difference that might have been present after the acute episode. Evidence exists to confirm this assumption, as shown by Svien

* Seven additional cases have been studied since this report was submitted. All seven were proved by arteriography or surgery and all showed significant retinal artery pressure differences. Four of the seven had incorrect diagnoses until ophthalmodynamometry was performed.

and Hollenhorst's case[8] in which the large difference in retinal artery pressures after surgical ligation of the carotid disappeared within one and one-half years, presumably as collateral circulation developed. It has been suggested that changes in retinal artery pressure following surgical ligation of the carotid serve as an index of the effectiveness of the ligation as well as an index of the subsequent development of collateral flow.[24, 26]

A typical illustrative case in this series of carotid artery thrombosis is summarized below:

E. B., a 65-year-old white woman, previously in good health, was hospitalized in August, 1957, for intermittent weakness of the left side. History also revealed some episodes of blurring in the right eye.

Neurologic examination showed impaired fine hand movements on the left and a left Babinski. Ophthalmologic examination revealed normal visual acuity, O.U., and full visual fields. A diagnosis of thrombosis of a small branch of the left middle cerebral artery was made and the patient discharged.

Four months later the patient suddenly developed left hemiparesis and aphasia. She had no further visual symptoms, and visual acuity was still normal; however, the patient had developed a left homonymous inferior quadrantic field defect. She was felt to have had a further thrombosis of the middle cerebral artery.

Ophthalmodynamometry was then done and the surprising finding of a markedly lower retinal artery pressure on the right was noted (systolic pressure 71 percent lower than O.S.; diastolic pressure 66 percent lower than O.S.). The question of carotid artery thrombosis was considered. Arteriography (fig. 6) was done, revealing a thrombosis of the right carotid artery two cm. above the bifurcation. The patient subsequently had surgery for removal of the thrombus from the carotid.

Two patients had the occlusion beyond the opening of the ophthalmic artery. The case of of A. H. is of particular interest as it presents an unusual problem for ophthalmodynamometry. We would not expect a lower retinal artery pressure on the side of the occlusion since, in this instance, the vascular pathway from the origin of the carotid to the ophthalmic is patent. Interestingly enough the pressure was significantly higher on the side of the occlusion. Basilar artery thrombosis can produce high carotid artery (thus retinal artery) pressures but this disease was ruled out by the arteriogram (fig. 7), which reveals the carotid block just above the ophthalmic artery branch. Observing the arteriogram it can be speculated that the entire head of carotid artery pressure was delivered di-

Fig. 6 (Spalter). E. B. Bilateral carotid arteriogram. Normal filling on left. Carotid occlusion on right. Dye seen in carotid siphon on right due to collateral flow from external carotid through ophthalmic artery in a retrograde manner. Retinal artery pressures: O.D., 30/20 mm. Hg; O.S., 105/60 mm. Hg.

Fig. 7 (Spalter). A. H. Right carotid arteriogram. Note flow through carotid is blocked just beyond ophthalmic artery opening. In serial films (four, five, and six seconds after dye injection) the ophthalmic artery (arrows) is well outlined and dilated. Retinal artery pressures: O.D., 110/55 mm. Hg; O.S., 70/30 mm. Hg.

rectly to the ophthalmic artery, resulting in a higher pressure reading by ophthalmodynamometry. Over the course of several weeks, the pressure on the occluded side slowly diminished becoming essentially equal to the contralateral side.

A case with carotid artery thrombosis beyond the ophthalmic artery opening with equal retinal artery pressure measurements has been reported[8] but I can find no cases in the literature with higher pressure on the side of the occlusion.

The most significant contribution of ophthalmodynamometry would be in its potential usefulness in determining which of the patients with nonspecific cerebral symptomatology should be suspected of carotid thrombosis. The average case presenting to the physician without a suggestive symptom complex (for example, intermittent amaurosis) must rest in a state of diagnostic confusion unless arteriography is done. However, because of the previously mentioned hazards associated with arteriography, the technique is not routinely used. If ophthalmodynamometry proves to be significantly reliable, it could be used to determine which cases should have arteriography; or perhaps it might even help one to dispense with the arteriogram and make a definitive diagnosis on the basis of positive ophthalmodynamometric findings. Van Allen and Blodi[24] have recently successfully utilized the technique in this manner.

Five of the 12 cases had retinal artery pressures determined prior to arteriography. In four of these, incorrect diagnoses had determined the therapeutic regimen until the positive findings with the ophthalmodynamometer indicated a carotid artery occlusion, subsequently confirmed by arteriography. All four of these patients then had operations for removal of the thrombosis, two successfully. In each instance it can be stated with fair certainty that without ophthalmodynamometry the cases would not have been subjected to arteriography and presumably the proper diagnosis would have been missed.

As a screening device ophthalmodynamometry appears to be fairly reliable in the relatively small series studied to date. Five cases were studied in which internal carotid artery thrombosis entered into the initial differential diagnosis (fig. 8). These cases

PATIENT	AGE	RETINAL ARTERY PRESSURE O.D.	O.S.	PER CENT DIFFERENCE	DIAGNOSIS	PROOF
M.L.	57	80/35	75/35	6/0	Meningioma	Surgery
H.P.	33	85/45	85/48	0/6	Astrocytoma	Surgery
T.K.	66	/110	/115	4	Tumor	Arteriogram
G.W.	58	/95	/105	10	Meningioma	Surgery
B.S.	52	82/30	80/32	2/6	A.V.Malformation	Arteriogram

Fig. 8 (Spalter). Ophthalmodynamometric findings in patients suspected of carotid artery thrombosis.

showed no significant differences in retinal artery pressure. Subsequently each was proven to have other disease entities. To date, there have been no falsely positive or falsely negative determinations. However, the series is small. Cases in which no significant difference is found by ophthalmodynamometry, suggesting the absence of carotid artery disease, may not be subjected to definitive diagnostic techniques, for example arteriography or surgery. In these situations then we are left with only strong clinical suspicions but no absolute proof that carotid thrombosis does not exist. As the number of reported cases increases in which ophthalmodynamometry has indicated the presence or absence of carotid disease (which is then subsequently proven or disproven) we can better evaluate the efficiency of the technique.

The recent introduction of direct surgical approaches to carotid thrombosis has introduced another area of usefulness for ophthalmodynamometry. At operation with direct visualization of the carotid artery the neurosurgeon is able to determine whether flow has been re-established following removal of the thrombosis. Postoperatively, however, maintenance of the vascular pathway can be determined only by clinical improvement which may be difficult to assess, or by repeat arteriography. Even if no clinical improvement occurs the physician cannot validly assess the technical success of the operative procedure since irreversible brain damage could have occurred prior to operation.

Of the five cases in this series that were operated upon, two showed a definite increase in retinal artery pressure on the side of the occlusion postoperatively so that the original pressure difference of 50 or more percent was reduced to less than five percent in each case. This equalization of pressure has been maintained until the time of this writing, three weeks and three months respectively. One of the cases, that of A. J., was particularly illustrative in that there was immediate clinical improvement as the retinal artery pressure increased postoperatively. A summary of the pertinent findings is found in Figure 9. A brief clinical summary follows:

A. J., a 58-year-old white man, was admitted to Neurological Institute in January, 1958, with a history of excellent health until 48 hours prior to admission when he suddenly lost vision in the left eye. Eight hours prior to admission he developed a right hemiplegia and aphasia. Previous neurologic or ophthalmologic symptoms were denied.

Neurologic examination revealed a severe right hemiparesis, a right Babinski, and expressive aphasia. Ophthalmologic examination revealed normal acuity and field, O.D., and barely light perception, O.S. Ophthalmodynamometry readings were: O.D., 130/70; O.S., 60/30.

Carotid thrombosis was then suspected and arteriography was done, revealing a block at the common carotid bifurcation on the left. The patient was operated on immediately, with removal of the thrombus and stripping of the intima of the artery. Good retrograde flow was established at operation.

Ten hours postoperatively the patient was able to recognize movement at one foot, and the retinal

VISUAL ACUITY O.S.	TIME OF OBSERVATION	OPHTHALMODYNAMOMETER O.D.	O.S.	% DIFFERENCE
L.P.	48 hours after onset of blindness O.S.	130/70	60/30	54%
	50 hours	Arteriogram: block at bifurcation of left common carotid artery		
	54 hours	Surgery: removal of thrombus; retrograde flow established		
H.M.	64 hours	/60	/40	33%
C.F. 1 foot	80 Hours	130/68	100/60	23%
C.F. 3feet	2 weeks	102/64	96/62	7%

Fig. 9 (Spalter). A. J. Summary of serial determinations of retinal artery pressures in patient with carotid artery thrombosis before and after successful surgery for thrombus removal.

artery diastolic pressures were: O.D., 60; O.S., 40, a difference of only 33 percent compared to the original difference of 55 percent (note the fall in absolute pressure, O.D., related to the fall in systemic blood pressure, postoperatively). Sixteen hours postoperatively the pressure difference had diminished to 23 percent. Two weeks postoperatively the patient was able to count fingers at three feet and dynamometry readings showed no significant difference. The patient is now beginning to show some neurologic improvement.

In addition to providing an immediate index of the technical success of the operative procedure a baseline measurement is obtained which is useful in following the patient over the years. If a patient with equalization of retinal artery pressures following a carotid thrombectomy suddenly develops progression of neurologic disease, a change in retinal artery pressure may provide an immediate index of recurrence of carotid thrombosis. Increasing retinal artery pressure on the side of the occlusion with or without surgery should also provide an index of the development of collaterals. The present cases have not yet been followed long enough to evaluate this potential use.

In reviewing the ocular findings of the 12 patients studied and an additional 18 others whose hospital records were available for review (fig. 10), it was interesting to note that four patients had intermittent amaurosis as their initial symptom of carotid artery disease. The visual field changes were nonspecific and appeared to reflect the varying vascular insult to the optic pathways. Pupillary changes were noted but were not helpful in diagnosis. No papilledema was seen in any of the patients, although others have seen this finding and attributed it to cerebral edema following the initial vascular episode. No cases showed any fundus change, in particular cotton-wool patches which have been found frequently in the ipsilateral eye by Hollenhorst.[28] The cases he reported in which this finding was noted had intermittent carotid insufficiency rather than complete occlusion, as in this series.

The classic concept of optic atrophy as the dominant ocular finding associated with carotid artery thrombosis was not borne out by this series. It is to be expected that where the thrombosis has extended into the intraorbital circulation and blocked all collaterals, optic atrophy might result; however, this situation is rare. Occasionally a central retinal artery occlusion can result secondary to embolization from the carotid artery thrombus, thereby eventually producing optic atrophy. In 10 cases measured, the intraocular pressure was normal and equal on both sides.

Of interest are the initial diagnoses recorded. In only a small percentage was the correct diagnosis entertained initially, demonstrating the difficulty in differential diagnosis in this disease.

Pathophysiology of Intermittent Amaurosis in Carotid Thrombosis

The pathophysiology of the predominant ocular symptom of intermittent amaurosis in this disease is an intriguing one and the finding of decreased retinal artery pressure on

I. OPHTHALMOLOGIC FINDINGS	
1) Visual Loss	
a) Intermittent Amaurosis	6 cases
b) Permanent Loss	1 case
c) As a presenting symptom	4 cases
2) Visual Fields	
a) Homonymous Hemianopia	4 cases
b) Quadrantic Defects	4 cases
c) Normal	14 cases
d) Not Done	8 cases
3) Pupillary Changes	3 cases
4) Diplopia (transient)	1 cases
5) Papilledema	0
6) Optic Atrophy	0
7) Fundus Changes (e.g. "cotton-wool" patches)	0
8) Tonometric Difference (Schiotz)	0 cases of 10 measured
9) Ophthalmodynanometric difference in occlusion proximal to ophthalmic artery	9 cases of 10 measured; tenth case measured 3 years after occlusion
II. INITIAL DIAGNOSIS	
1) Brain Tumor	13 cases
2) Middle Cerebral Artery Thrombosis	7 cases
3) Carotid Artery Thrombosis	4 cases
4) Aneurysm	1 case
5) Idiopathic Epilepsy	1 case
6) Deferred or " Unknown "	4 cases

Fig. 10 (Spalter). Summary of 30 cases of carotid artery thrombosis proved by arteriography or surgery.

the side of the occlusion leads to some interesting speculation. At first it would seem likely that a simple valid explanation is that intermittent occlusion of an already severely narrowed carotid artery would produce intermittent visual and neurologic disturbances. However, it is well known that surgical ligation of the common or internal carotid artery is rarely accompanied by visual loss.

In the classic experiments of Walsh and King[29] in 1942, the enormous collateral circulation potential of the eye was first accurately demonstrated. This work offered the explanation as to why the visual function escapes damage despite obstruction of the eye's main feeding vessel, the carotid. In a series of perfusion experiments they demonstrated that with the internal carotid occluded, the ophthalmic artery fills by way of the collaterals from the external carotid of the same side. In 1956, Shea[30] repeated these experiments and demonstrated anastomoses with the external carotid circulation on the same side in 91 percent of 49 cadavers studied and 31 percent with collaterals from the contralateral external carotid. These authors worked with perfusion pressures well within the physiologic range. It is also to be expected that the internal carotid from the contralateral side aids in the ophthalmic circulation through the circle of Willis. This can be demonstrated arteriographically when the patent carotid is injected and the dye crosses over to the side of the occlusion.

It would seem, therefore, that collateral flow adequately reaches the ophthalmic artery to supply the retina in occlusion of the carotid. In the series of 30 cases in this study 60 percent had demonstrable visualization of the ophthalmic artery by collateral flow

from the external carotid in a retrograde manner (fig. 6). I was unable to correlate the presence or absence of visual symptoms with the presence of retrograde flow and I believe that collateral circulation can be assumed in all cases, whether demonstrated by the gross technique of X rays or not. François'[31] study of the optic nerve blood supply by microarteriography reveals the vast number of collateral channels available between the external and internal carotid supply to the eye and is impressive evidence that carotid artery obstruction per se should not inhibit retinal function. In general, then, we can discount carotid obstruction itself as the direct cause of the visual symptoms, except in those rare instances where the thrombus extends to the origin of the central retinal artery or has thrown an embolus to that side, and even in this situation we would expect constant rather than intermittent blindness, as well as funduscopic evidence of ischemia.

A second consideration to explain this symptom would be vasospasm. There is much controversy about whether this exists at all in the intracerebral circulation. It is known that cervical sympathectomy will not abolish the visual or neurologic symptoms in carotid disease. It is also to be questioned whether a narrowed vessel represents primary vasospasm or is a reflex narrowing secondary to a change in blood pressure. Also, if vasospasm is a factor, it would have to affect selectively the retinal vessels and cerebral vessels at different times, as the intermittent visual and neurologic symptoms in most instances do not occur together.

Perhaps the most attractive hypothesis is that the symptoms are due to transient falls in systemic blood pressure—a fall due either to postural change, emotional stress, or any one of a variety of causes of blood-pressure change that would decrease blood flow sufficiently to compromise function. We are dealing with areas, whether retina or cerebral cortex, that are already working at a disadvantage of collateral supply alone, with a decreased arterial pressure head as shown by ophthalmodynamometry or direct intracarotid pressure measurements. Thus the supply of blood may be just sufficient to support function or with a minimal reserve. Any further decrease in flow because of a fall in blood pressure could then make itself manifest symptomatically in a decrease in function, such as visual loss. The efficiency of collateral circulation must vary from area to area, depending upon the number of collaterals, their patency, and the metabolic demands of the area they serve. A small drop in blood pressure may thus affect only vision, whereas a larger drop is required to compromise some cerebral function, or vice versa, thereby offering an explanation for the shifting symptomatology.

Clinical support for this hypothesis may be found in a recent case report.[32] A patient with proven bilateral carotid artery disease was asymptomatic, with a systemic blood pressure above 160 mm. Hg. When the pressure fell below this value because of postural change or was induced by drugs, the patient consistently developed mental confusion and paresis of the extremities. The symptoms would then clear when the pressure returned to 160 mm. Hg.

Experimental support is found in the work of Meyer and co-workers[33] who, in 1954, showed that the brain damage in Rhesus monkeys following carotid occlusion varied according to the presence of adequate collaterals at autopsy. Measuring the animals in vivo they demonstrated by the polographic method of recording oxygen availability on the surface of the brain that the adequacy of collateral circulation, and thus brain function, depended upon the maintenance of systemic blood pressure.

Webster,[34] in 1957, postulated that the vessels of the meninges, in particular the pial vessels, play a large role in collateral circulation of the brain. In animal experiments enlargement of these vessels is clearly shown following proximal occlusion. The

enlarged pial vessels are then shown to collapse following an induced drop in systemic blood pressure. It seems likely that the same situation pertains to the collateral vessels supplying the ophthalmic artery (and the central retinal artery), perhaps explaining the transient nature of the visual loss.

It would seem that the entity of carotid artery disease can provide excellent clinical and experimental material for further studies on retinal function in relation to vascular supply. Several authors have recently suggested a correlation between flicker fusion field changes and retinal blood flow. It would be of interest to utilize this test in an individual with carotid artery disease with decreased retinal artery pressure. Here one can assume a unilaterally reduced blood supply and then compare the fields in that eye to the fields in the contralateral eye with its normal blood supply. Similarly the electroretinogram might reveal selective differences and add further insight into this problem.

Summary

1. Fifty normal subjects were found to have reproducible retinal artery measurements by ophthalmodynamometry. The difference in pressure between the two eyes of any one individual did not exceed 15 percent.

2. Twelve patients with carotid artery thrombosis were measured with Bailliart's ophthalmodynamometer. Of the 10 cases that would be expected to reveal a difference, nine had significant differences in retinal artery pressure ranging from 29 to 86 percent lower on the side of the occlusion. The 10th case was measured three years after occlusion and is presumed to have developed collateral flow sufficient to equalize pressures. Two patients had the occlusion distal to the ophthalmic artery opening, and one of these had a significantly higher retinal artery pressure on the side of the occlusion.

3. In four cases the diagnosis was not apparent until ophthalmodynamometric differences were demonstrated.

4. The usefulness of retinal artery pressure measurements following surgery for carotid artery thrombosis is illustrated and discussed.

5. Five patients suspected of carotid artery disease with no significant retinal artery pressure difference were subsequently proven not to have carotid artery disease.

6. The ocular findings in a series of 30 proven cases of carotid artery thrombosis are presented and the pathophysiology of the predominant ocular symptom of intermittent amaurosis is discussed.

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References

1. Donders, F. C.: Ueber die sichtbaren Erschainungen der Blutbewegung im Auge. Arch. f. Ophth., 1:75-105, 1854-1855.

2. Bailliart, P.: La pression artérielle dans les branches de l'artére centrale de la rétine: Nouvelle technique pour la déterminer. Ann. ocul., **154**:648-666 (Nov.) 1917.

3. ———: The Retinal Circulation. Chicago, Professional Press, Inc., 1928.

4. Smith, P.: The blood pressure in he eye and its relation to the chamber pressure. Brit. J. Ophth., **1**:4-26, 657-677 (Jan., Nov.) 1917; **7**:449-469 (Oct.) 1923.

5. Duke-Elder, W. S.: The ocular circulation: Its normal pressure relationships and their physiological significance. Brit. J. Ophth., **10**:513-572 (Oct.) 1926.

6. Koch, F. L. P.: Ophthalmodynamometry. Arch. Ophth., **34**:234-247 (Sept.) 1945.

7. De la Fuente, G. L., and Sanchez, R. B.: Modificaciones de la presion arterial retiniana tras la compresion de la carotida primitiva. Arch. Soc. oftal hispano-am., **8**:457-467 (May) 1948.

8. Svien, H. J., and Hollenhorst, R. W.: Pressure in retinal arteries after ligation or occlusion of the carotid artery. Proc. Staff Meet. Mayo Clin., **31**:684-692 (Dec.) 1956.

9. Thomas, M. H., and Petrohelos, M. A.: Diagnostic significance of retinal artery pressure in internal carotid involvement: findings in eight cases with carotid-artery lesions compared to 50 controls and 210 cases from literature. Am. J. Ophth., **36**:335-346 (Mar.) 1953.

10. Wood, F. A., and Toole, J. F.: Carotid occlusion and its diagnosis by ophthalmodynamometry. J.A.M.A., **165**:1264-1270 (Nov.) 1957.

11. (a) Fisher, M.: Occlusion of the internal carotid artery. Arch. Neurol. & Psychiat., **65**:346-377 (Mar.) 1951; (b) Occlusion of the carotid arteries: Further experiences. Arch. Neurol. & Psychiat., **72**:187-204 (Aug.) 1952.

12. Boyd, J. D.: Absence of the right common carotid artery. J. Anat., **68**:551-557 (July) 1934.

13. Duffy, P. E.: Portnoy, B., Mauro, J., and Wehrle, D. F.: Acute infantile hemiplegia secondary to spontaneous carotid thrombosis. Neurology, **7**:664-666 (Aug.) 1957.

14. Johnson, H. C., and Walker, J.: The angiographic diagnosis of spontaneous thrombosis of the internal and common carotid. J. Neurosurg., **8**:631-659 (Nov.) 1951.

15. Eastcott, H. H. G., Pickering, G. W., and Rob, C. G.: Reconstruction of internal carotid artery in a patient with intermittent attacks of hemiplegia. Lancet, **2**:994-996 (Nov.) 1954.

16. Strully, K. J., Hurwitt, E. S., and Blankenberg, H. W.: Thrombo-endarterectomy for thrombosis of the internal carotid artery in the neck. J. Neurosurg., **10**:474-482 (Sept.) 1953.

17. Van Allen, M. W., Blodi, F. C., and Brintnall, E. S.: Retinal artery blood pressure measurements in diagnosis and surgery of spontaneous carotid occlusions. J. Neurosurg., **15**:19-29 (Jan.) 1958.

18. Moniz, E., Lima, A., and De Lacerda, R.: Hemiplegies par thrombose de la carotide artere. Presse med., **45**:977-980 (June) 1937.

19. Walsh, F. B., and Smith, G. W.: The ocular complications of carotid angiography: The ocular signs of thrombosis of the internal carotid artery. J. Neurosurg. **9**:517-537 (Sept.) 1952.

20. Rosegay, H.: Limited value of carotid pulse in the diagnosis of internal carotid artery thrombosis. Neurology, **6**:142-145 (Feb.) 1956.

21. Meyer, J. S., Leiderman, H., and Denny-Brown, D.: Electroencephalographic study of insufficiency of the basilar and carotid arteries in man. Neurology, **6**:455-477 (July) 1956.

22. Sweet, W. H., and Bennett, H. S.: Changes in internal carotid pressure during carotid and jugular occlusion and their clinical significance. J. Neurosurg., **5**:178-195 (Mar.) 1948.

23. Bakay, L., and Sweet, W. H.: Cervical and intracranial intra-arterial pressures with and without vascular occlusion. Surg. Gyn. & Obst., **95**:67-75 (July) 1952.

24. Van Allen, M. W., and Blodi, F.: Retinal artery blood pressure changes (with compression or ligation of the carotid arteries). Am. J. Ophth., **45**:364-371 (Mar.) 1958.

25. Miletti, M.: Le diagnostic de la thrombose primitive de la carotide interne dans la region cervicale au moyen de la determination des valeurs de la pression systolique de l'artere centrale de la retine. Presse md., **54**:655-656 (Oct.) 1946.

26. Heyman, A., Karp, H. R., and Bloor, B. M.: Determination of retinal artery pressures in diagnosis of carotid artery occlusion. Neurology, **7**:97-104 (Feb.) 1957.

27. Weigelin, E., Althaus, H., Bernhard, C., Reckmann, P., and Ossendorff, I.: Beurteilung des intracraneillen Kreislaufs mit Hilfe der Netzhautarteriendruckmessung. Documenta Ophth., **7-8**:183-245, 1954.

28. Hollenhorst, R. W.: Am. J. Ophth. **44**:829-832 (Dec.) 1957.

29. Walsh, F. B., and King, A. B.: Ocular signs of intracranial saccular aneurysms: Experimental work on collateral circulation through the ophthalmic artery. Arch. Ophth., **27**:1-33 (Jan.) 1942.

30. Shea, M.: Carotid-ophthalmic anastomoses: Frequency of external carotid and ophthalmic artery anastomoses. Brit. J. Ophth., **40**:497-501 (Aug.) 1956.

31. François, J., and Neetens, A.: Vascularization of the optic pathway. Brit. J. Ophth., **38**:472-488 (Aug.) 1952.

32. Shanbrom, E., and Levy, L.: The role of systemic blood pressure in cerebral circulation in carotid and basilar artery thromboses: Clinical observations and therapeutic implications of vasopressor agents. Am. J. Med., **23**:197-204 (Aug.) 1957.

33. Meyer, J. S., Fang, H. C., and Denny-Brown, D.: Polarographic study of cerebral collateral circulation. Arch. Neurol. & Psych., **72**:296-312 (Sept.) 1954.

34. Webster, J. E., Gurdjian, E. S., and Martin, F. A.: Considerations concerning increasing cerebral blood flow in cerebral insufficiency. J. Neurosurg., **14**:152-159 (Mar.) 1957.

ANISEIKONIA FOLLOWING RETINAL DETACHMENT*

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As a consequence of the great technical advances recently made in the treatment of retinal detachment, the visual rehabilitation of these patients is becoming an increasing problem. Retinal detachment and its repair often reduce visual acuity and field. Retinal degeneration, notably in the macula area, is the most frequent cause; but opacities of the media, both of the vitreous and the lens, may contribute to the visual loss.

In addition to such monocular problems, binocular efficiency may be seriously impaired by the postoperative onset of heterophorias, especially vertical, which result from the involvement of the extraocular muscles in the surgical repair.

It is the purpose of this paper to report another entity capable of greatly reducing binocular efficiency in this group of patients, namely, aniseikonia. Eyes which have undergone retinal detachment and subsequent repair almost unfailingly demonstrate a diminution of image size.

A small amount of aniseikonia (physiologic) is not considered abnormal and the visual mechanism easily compensates for it. Furthermore, it is essential for stereopsis. Greater degrees of image size difference (anomalous aniseikonia) are capable of producing severe asthenopic symptoms and even a complete breakdown in binocular vision (Burian[1] 1943). An over-all image size difference of one percent is presently considered significant. Differences of three percent produce definite impairment of function and, at five percent, binocular vision is either absent under normal condition or imperfect. The symptoms vary from case to case, depending upon the amount, the type of image change, and the individual's propensity for psychologic adaptation and physiologic compensation (Burian,[2] 1943).

* From the Department of Ophthalmology, Manhattan Eye, Ear, and Throat Hospital. Presented at the IV Interim Congress of the Pan-American Association of Ophthalmology, April, 1957, New York.

METHOD AND RESULTS

The patients selected for this study were limited to those with cases of serous retinal detachment who had undergone surgery with subsequent reattachment. Several had more than one operation. In all cases only scleral diathermy was used. Penetrating, partially penetrating, or surface diathermy was used. Inasmuch as the vision of both eyes must be of a sufficient degree to permit eikonometer evaluation, no patient with a corrected vision in either eye of less than 20/70 was included in the study. Both the space and standard eikonometers were used.

Twelve such patients have been studied and in all cases an anomalous aniseikonia could be demonstrated. The difference in image size was over-all in type, with additional meridional discrepancies. In all but one case there was minification of the retinal image in the operated eye. In Case 8 there was a minification of the image in the unoperated eye of anomalous degree, a finding which suggests the possibility of a preexistent aniseikonia.

REPORT OF CASES

CASE 1

R. McE., a boy, aged 13 years, was admitted on November 29, 1951, with the diagnosis of detachment of retina, O.S., four or five months in duration. The macula was not detached.

Surgery on December 5th: Partial penetrating diathermy at the 4- to 8-o'clock positions. On February 29, 1952: Partial penetrating diathermy at the 11- to 1-o'clock positions. Vision: O.D., plano = 20/20; O.S., plano = 20/50.

Aniseikonia examination: Diminution of retinal image, O.S.; vertical meridian, one percent; horizontal meridian, two percent.

CASE 2

M. H., a man, aged 57 years, was admitted November 5, 1951, with the diagnosis of detachment of

retina, O.S., of 10 weeks' duration. The macula was detached.

Surgery on November 10, 1951: Penetrating diathermy at the 10- to 12-o'clock positions. Vision: O.D., +0.75D. sph. ⌒ −1.0D. cyl. ax. 180° = 20/20; O.S., +0.25D. sph. ⌒ −1.75D. cyl. ax. 180° = 20/60.

Aniseikonia examination: Diminution of retinal image, O.S.; vertical meridian, five percent; horizontal meridian, three percent.

CASE 3

S. G., a man, aged 62 years, was admitted on November 20, 1951, with the diagnosis of detachment of retina, O.S., of four weeks' duration. The macula was not detached.

Surgery on November 24, 1951: Penetrating and surface diathermy from the 7:30- to 10:30-o'clock positions. Vision: O.D., +0.75D. sph. ⌒ −0.75D. cyl. ax. 175° = 20/20; O.S., +0.50D. sph. ⌒ −0.50D. cyl. ax. 115° = 20/25.

Aniseikonia examination: Diminution of retinal image, O.S.; vertical meridian, ? percent; horizontal meridian, 2.5 percent.

CASE 4

M. G., a woman, aged 41 years, was admitted on November 8, 1951, with a diagnosis of detachment of retina, O.D., of eight weeks' duration. Macula detached (probably).

At surgery on December 19, 1951, penetrating diathermy was done at the 7- to 11-o'clock positions and on April 1, 1952, at the 9- to 2-o'clock positions with a saline implant. Vision was: O.D., plano = 20/70; O.S., plano = 20/40.

Aniseikonia examination: diminution of retinal image, O.D., vertical meridian, 1.5 percent; horizontal meridian, one percent.

CASE 5

T. B., a man, aged 64 years, was admitted on July 5, 1950, with a diagnosis of detachment of retina, O.D., duration undetermined. The macula was not detached (probably). At surgery on August 4, 1950, penetrating diathermy was done at the 10- to 11-o'clock positions and on December 7th from the 10- to 11-o'clock positions. Vision: O.D., +1.0D. sph. ⌒ −1.5D. cyl. ax. 95° = 20/30 (2Δ base-up); O.S., +0.75D. sph. ⌒ −1.25D. cyl. ax. 95° = 20/25 (2Δ base-down).

Aniseikonia examination: diminution of retinal image, O.D., vertical meridian, 2.5 percent; horizontal meridian, five percent plus. This patient is wearing aniseikonic correction with relief of asthenopia.

CASE 6

E. C., a man, aged 54 years, was admitted on October 2, 1954, with a diagnosis of detachment of retina, O.S., of eight weeks' duration. The macula was not detached.

Surgery on October 7, 1954: Penetrating, partial penetrating, and surface diathermy from the 4- to 6-o'clock positions. Vision: O.D., −0.75D. sph. = 20/20; O.S., −2.25D. sph. = 20/20−.

Aniseikonia examination: diminution of retinal image, O.S., vertical meridian, ? per cent; horizontal meridian, three percent.

CASE 7

T. B., a woman, aged 27 years, was admitted on February 17, 1945, with a diagnosis of detachment of retina, O.D., of undetermined duration. The macula was not detached. In 1940, the detachment of retina, O.S., was repaired with diathermy; in 1941, detachment of retina, O.D., was repaired with diathermy.

At surgery on February 26, 1945, penetrating diathermy was done at the 1-o'clock position. Vision was: O.D., −8.25D. sph. ⌒ −2.50D. cyl. ax. 15° = 20/25; O.S., −9.00D. sph. ⌒ −3.50D. cyl. ax. 163° = 20/30.

Aniseikonia examination: Diminution of retinal image, O.D., vertical meridian, 3.5 percent; horizontal meridian, 2.0 percent. The patient is wearing aniseikonic correction with relief of asthenopia.

CASE 8

H. S., a woman, aged 58 years, was admitted on September 5, 1954, with a diagnosis of detachment of retina, O.D., of four weeks' duration. The macula was not detached.

At surgery on September 23, 1954, penetrating, partial penetrating, and surface diathermy was done at the 10- to 12-o'clock positions. Vision was: O.D., plano = 20/20; O.S. +1.0D. sph. ⌒ −0.5D. cyl. ax. 90° = 20/60.

Aniseikonia examination: Diminution of retinal image, O.S., vertical meridian 1.5 percent; horizontal meridian, 2.5 percent.

CASE 9

H. S., a woman, aged 52 years, was admitted on August 5, 1954, with a diagnosis of detachment of retina, O.D., of one week's duration. The macula detachment was undetermined.

At surgery on September 6, 1954, penetrating and surface diathermy was done at the 7- to 10-o'clock position, with air injection. Vision was: O.D., +0.25D. sph. ⌒ −1.0D. cyl. ax. 80° = 20/50; O.S., +0.25D. sph. ⌒ −1.0D. cyl. ax. 90° = 20/20.

Aniseikonia examination: Diminution of retinal image, O.D., vertical meridian, five to six percent; horizontal meridian, four percent.

CASE 10

R. B., a man, aged 18 years, was admitted on April 23, 1954, with a diagnosis of detachment of retina, O.D., of one week's duration. The macula was not detached.

At surgery on May 3, 1954, partial penetrating diathermy was done at the 10- to 2-o'clock position. Vision was: O.D., −0.50D. cyl. ax. 45° = 20/30; O.S., +0.25D. sph. ⌒ −0.5D. cyl. ax. 45° = 20/30.

Aniseikonia examination: Diminution of retinal image, O.D., 160-degree meridian, two percent.

CASE 11

J. McQ., a boy, aged 15 years, was admitted on November 15, 1952, with a diagnosis of detachment

of retina, O.D., of two weeks' duration. The macula detachment was undetermined.

At surgery on November 21, 1952, penetrating and surface diathermy was done at the 11-o'clock position; December 8, 1952, penetrating and partial penetrating diathermy, at the 11- to 2-o'clock position; January 2, 1953, penetrating diathermy at the 9- to 1-o'clock position; February 11, 1953, partial penetrating diathermy at the 5- to 7-o'clock position with vitreous implant. Vision was: O.D., −1.25D. sph. ⊂ −0.5D. cyl. ax. 15° =20/25−; O.S., −0.12D. sph. ⊂ −0.5D. cyl. ax. 15° = 20/20.

Aniseikonia examination: Diminution of retinal image, O.D., standard eikonometer, vertical meridian, 3.5 percent; horizontal meridian, 6.0 percent. Space eikonometer, vertical meridian, 3.0 percent; horizontal meridian, 8.0 percent.

CASE 12

L. I., a man, aged 59 years, was admitted on October 29, 1956, with a diagnosis of detachment of retina, O.S., of two weeks' duration. The macula was not detached. Vision was: O.D., +2.0D. sph. ⊂ −0.75D. cyl. ax. 5° = 20/20; O.S., +2.0D. sph. ⊂ −0.75D. cyl. ax. 5° = 20/25−1.

Aniseikonia examination: Diminution of retinal image, O.S., vertical meridian, 0.5 percent; horizontal meridian, 0.0 percent.

At surgery on November 5, 1956, partial-penetrating diathermy was done at the 9:30- to 11.30-o'clock positions with vitreous implant. Vision was: O.D., +2.0D. sph. ⊂ −0.75D. cyl. ax. 5° = 20/20; O.S., −0.5D. cyl. ax. 135° = 20/20.

Aniseikonia examination, January 9, 1957: Diminution of retinal image, O.S., vertical meridian, 0.0 percent; horizontal meridian, 2.0 percent. Aniseikonia examination, March 15, 1957: Diminution of retinal image, O.S., vertical meridian 0.5 percent; horizontal meridian, 0.5 percent.

DISCUSSION

Minification of the retinal image may be produced, optically, by the movement of the retina closer to the second nodal point, or, anatomically, by the separation of the neuroepithelial elements in the outer retina (Ames, Gliddon, and Ogle, 1932).

Scleral shortening procedures can substantially reduce the axial length of the globe and thereby produce an optical aniseikonia. It is not likely that simple diathermy is capable of a similar effect. Diathermy applications do effect a certain amount of scleral shrinkage but any decrease in axial length as a result is generally conceded to be minimal. The absence of significant changes in refraction following such treatment argues against such a mechanism.

Acquired anatomic aniseikonia may result from retinal edema. Alterations in the normal extracellular fluid interchange of the outer layers of the detached retina may result from the loss of blood supply. The trauma of surgical repair must also be considered as a cause of retinal edema as well as the more remote possibilities of hypotony and the toxic effects of the subretinal fluids.

Another possible mechanism of acquired anatomic aniseikonia is a stretching of the reattached retina. The temporary separation of the retinal cells by either mechanism may be made permanent later by organization of intercellular fluids and glial proliferation.

With the exception of Case 12, it is impossible to eliminate the question of preexistent aniseikonia. In this regard it is of interest to note that, in this series, the clinical symptoms of image distortion and fusional difficulties had their onset following retinal detachment. This would strongly suggest an acquired aniseikonia. Furthermore, the mathematical possibility of a random group of 12 patients all displaying anomalous aniseikonia up to eight percent is, at best, remote. On the other hand it is questionable that the statistics concerning aniseikonia can be validly applied to detachment patients as a group.

Striking anatomic changes both in the detached and fellow eye have been known for some time. Added to this is the recent abundant evidence of equally great physiologic changes as demonstrated by electroretinography (Karpe and Rendahl, 1952; Burian, 1953; François and de Rouck, 1953, and Rendahl, 1957).

In Case 12 both preoperative and postoperative studies were performed and, if this case may be considered typical, the image minification is effected by the surgical repair. The subsequent change in image measurements may be explained by the reabsorption of intercellular fluids or on the basis of Burian's psychologic adaptation and physiologic compensation.

The subject will need a great deal more

study before the etiologic mechanism and the nature and scope of the patient's adjustment are fully appreciated.

Conclusion

In spite of the limited scope of this study, aniseikonia would appear to be a common effect of retinal detachment treated by diathermy. The image size differences are of significant amounts and capable of producing extreme asthenopia and greatly reduced binocular efficiency. Patients who have undergone successful retinal surgery should be considered suspect of this condition especially if the symptomatology and vergences indicate a subnormal fusion range. These patients can be rehabilitated to greatly improved binocular vision through the use of aniseikonic corrections.

2488 Grand Concourse (58).

References

Ames, A., Gliddon, G., and Ogle, K.: Size and shape of ocular images; Methods of determination and physiologic significance. Arch. Ophth., **7**:576, 1932.

Burian, H.: Clinical significance of aniseikonia. Arch. Ophth., **29**:116, 1943; Influence of wearing of meridional size lenses on spatial localization. Arch. Ophth., **30**:645, 1943.

François, J., and de Rouck, A.: L'electroretinographie dans la myopie et les decollements myopigenes de la retina. Ophthalmologica, **33**:131, 1955.

Karpe, G., and Rendahl, I.. The clinical electroretinogram: VI. The electroretinogram in detachment of the retina. Ophthalmologica, **30**:303, 1952.

Rendahl, I.: The electroretinogram in detachment of the retina. Arch. Ophth., **57**:566, 1957.

Burian, H.: The electroretinogram and its clinical applications. Arch. Ophth., **49**:241, 1953.

OCULAR CHANGES IN PHEOCHROMOCYTOMA*

With a note on its mimicry of optic neuritis

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At first glance, the ophthalmologist might seem to be invading medical territory not in his own domain when he writes of pheochromocytoma. This is not true. Pheochromocytoma is a neoplasm which is, in many respects, equivalent to a systemic disease. It affects the eyes as it progresses. Without proper treatment, it may lead to death. It may also lead to blindness which is irreversible. The tragedy of both death and blindness caused by pheochromocytoma is that neither is inevitable. This is a curable disease, and the visual impairment which it may cause is also curable, if the diagnosis is made and if surgery is undertaken before it is too late.

Pheochromocytoma, as DeCourcy and DeCourcy[1] well express it, is "the great mimic." The constitutional disease with which it is most likely to be associated is hypertensive vascular disease. There is no doubt that at the present time some patients with pheochromocytoma are losing their lives because their disease is diagnosed as cardiovascular. The confusion would promptly be ended if all patients with hypertensive vascular disease were screened by the Regitine (Methanesulfonate†) test, a simple and extremely efficient method of differentiating the two conditions.

Cases of hypertensive vascular disease which progress to the malignant stage are always characterized by ocular changes, including choked discs and retinal edema, hemorrhages, and exudates. These changes

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† This is an adrenolytic drug which is thought to block the receptors of the sympathetic nerve impulses.

are not distinguishable by clinical methods from the ocular changes which may accompany pheochromocytoma. Attention has already been called to these changes,[2-4] though not with sufficient emphasis. There is, however, another possible source of diagnostic confusion arising from an associated process, the possibility—and from personal experience I know that it is a real possibility—of confusing the ocular involvement of pheochromocytoma with optic neuritis. As far as I can determine, no attention has previously been called to this situation, which has obvious serious and tragic implications.

There is, at the present time, no generally satisfactory treatment for optic neuritis. Many patients recover their vision but some progress to optic atrophy, with partial or total blindness. To misdiagnose the ocular involvement of pheochromocytoma as optic neuritis could be tragic from the standpoint of vision and fatal from the standpoint of survival. Any patient, therefore, who appears to have optic neuritis should, like patients with apparent hypertensive vascular disease, be carefully investigated with the possibility of pheochromocytoma in mind.

A normal blood pressure at the time of the ophthalmologic examination does not exclude the diagnosis of pheochromocytoma in a patient with apparent optic neuritis. In the following case, which is the basis of this communication, the blood pressure was within the limits of normal the day the patient was first seen but, 24 hours later, it was 100 points higher.

Case history

A 14-year-old white boy was referred for consultation December 30, 1954, by the ophthalmologist whom he had consulted because of blurred vision which had been present for two months. Over this time his sight had been slowly deteriorating but he had done nothing about it until it reached the point of interference with his school work. The visual difficulty was associated with a headache which was persistent but not very annoying. On December 20th, while on a hunting trip, he was unable to see the birds which his companions reported, and at a Christmas party he found he could not read the names on his gifts.

Fig. 1 (Gaines). Left fundus of patient with pheochromocytoma. Note resemblance of changes to those encountered in optic neuritis.

On questioning, he stated that about six months before this consultation he had noticed that his heart pounded more than usual during exercise and that he occasionally felt dizzy. Examination for a boys' camp in July, 1954, had revealed no abnormalities. On one occasion during the camp he had suffered from abdominal cramps; no specific cause was found for them. The blood pressure at this time was said to be normal, as it had been three months earlier (in March, 1954), when he was examined for a possible concussion.

The child was well developed and appeared normal in all respects. His blood pressure was 130/80 mm. Hg. Positive findings were confined to the eyes (figs. 1 and 2).

Vision in both eyes was limited to ability to count fingers at four feet. The pupils were equal and reacted to light and convergence. Corneal sensitivity was bilaterally equal. The disc in the right eye was markedly elevated and there were a few flame-shaped hemorrhages about it. The macular area was also elevated, and the area between the macula and the disc was extremely edematous. There was no notable spasm of the retinal arteries, and the difference in the size between the arteries and veins was considered to be due to venostasis. A beginning macular star was present. The findings in the left eye were essentially the same.

Peripheral visual fields (fig. 3) were full. A small central scotoma was present in each eye. Although fixation was poor, the outline of the field defect was definite. There was no paresis of the third, fourth, or sixth motor nerves.

A tentative diagnosis of optic neuritis was made on the basis of these findings, and neurologic consultation was requested.

Roentgenograms of the chest and skull showed

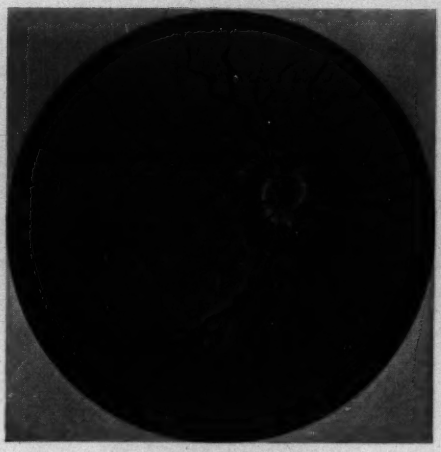

Fig. 2 (Gaines). Right fundus.

no abnormalities, and preparations for spinal tap were made. The child was quite tense during the positioning, and his blood pressure was found to be 210/60 mm. Hg in the right arm and 230/170 mm. Hg in the right leg. The spinal pressure was 270 mm. H_2O. In view of the subsequent progress in this case, the neurosurgeon thought, in retrospect, that the high pressure was due to the tense state of the child at the time of the examination and not to increased intracranial pressure.

At this point an internist was asked to see the patient. The detailed physical examination was essentially negative except for sinus tachycardia and a slightly enlarged heart. Abnormal changes were reported in the electrocardiogram but no specific diagnosis was made.

The blood Wassermann reaction was negative. Urinalysis showed one-plus albumin, which was still present five days later. The specific gravity was 1.021. There were occasional pus cells and very occasional red blood cells. The red blood cell count was 5,270,000 and the white blood cell count 15,800 per c. mm. The hemoglobin value was 13.5 gm. percent. The differential white blood cell count was within normal range. Blood chemisty determinations showed the following values: glucose 121 mg. percent; nonprotein nitrogen 39.5 mg. percent; uric acid 4.5 mg. percent. The basal metabolism rate was minus four.

The day after the spinal tap the patient suddenly became pale, cold, and clammy, though he remained oriented and responsive. The nurse was unable to get the blood pressure at all, but the interne, by palpation, found it to be 180 mm. Hg systolic. The pulse was 140 per minute.

The following day (January 2nd), while the patient was in nearly normotensive state (blood pressure 140/120 to 150/124 mm. Hg), the abdomen was palpated. The blood pressure promptly rose to 220/120 mm. Hg, then returned to the base line within four minutes. The same elevation occurred regardless of which side of the abdomen was palpated. No masses were felt. Later the same day, when the pressure was 190/160 mm. Hg, Regitine (three mg.) was injected intramuscularly. Within three minutes the pressure began to fall, and at the end of 30 minutes it was 130/102 mm. Hg.

Intravenous pyelography January 3rd revealed no regional abnormalities of any kind. Repetition of the examination after air insufflation of the retroperitoneal space showed a suprarenal mass, about five cm. in diameter, on the right side. This observation confirmed the diagnosis of pheochromocytoma, which had been entertained since the elevated blood pressure found at the spinal tap 24 hours after the pressure had been within normal range.

January 7, 1955, under general anesthesia (Pentothal sodium, ether, and ethylene), exploration of the right renal area was carried out. At the beginning of induction, the blood pressure was 240/180 mm. Hg. Within 15 minutes it had fallen to 170/140 mm. Hg. A tumor was palpated at the upper pole of the kidney. Palpation produced no change in the pressure, but when the renal pedicle was tied, the pressure fell to 100/60 mm. Hg. It rose promptly when Levophed bitartrate (1-Arterenol; 1-Norepinephrine) was administered and was maintained thereafter at about 170/140 mm. Hg. The right adrenal gland was resected without further difficulty and the child was in good condition at the conclusion of the operation.

The diagnosis of pheochromocytoma was confirmed in the laboratory. Pathologic details are omitted as not relevant to this report but illustrations of the gross microscopic pathology (figs. 4 to 7) are included to complete the record.

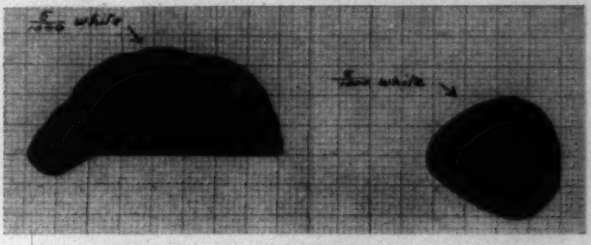

Fig. 3 (Gaines). Preoperative visual fields, December 30, 1954.

Fig. 4 (Gaines). Pheochromocytoma removed at operation from right adrenal gland.

Levophed was continued for the first 24 hours after operation. The blood pressure remained in the neighborhood of 126/84 mm. Hg. Recovery was without incident. The electrocardiogram taken on the 12th postoperative day was definitely improved over the preoperative electrocardiogram and was interpreted as possibly within normal limits. There was only a faint trace of albumin in the urine. When the child was discharged on the 14th postoperative day, the blood pressure was 120/60 mm. Hg.

He was kept under observation of his local physician but had no active treatment other than the intramuscular administration of vitamin B_{12} (1,000 μg.) twice weekly.

There had been no significant changes in the fundus picture during the patient's immediate postoperative period and his vision continued at about the preoperative level (ability to count fingers at four feet). Some slight objective improvement was observed on the day of his discharge from the hospital. Examination on March 2, 1955, showed no substantial changes in the eyes. The blood pressure at this time was 124/70 mm. Hg and the boy was in excellent condition, with no symptoms.

Fig. 5 (Gaines). Partially dissected specimen.

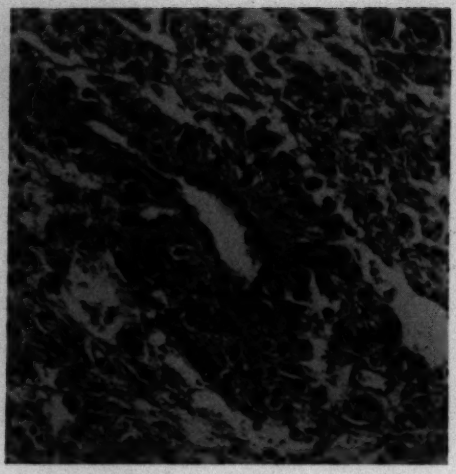

Fig. 6 (Gaines). Photomicrograph of tumor (low power).

At examination on April 13th, vision had improved to 20/50 in the right eye and 20/200 in the left. There was a very trivial error of refraction, but vision was not improved by the use of a +0.5D. sph. and a +0.5D. cyl. ax. 90° in both eyes. On funduscopic examination, some slight pallor of the discs was observed, as well as a few areas of hard exudate scattered about the retina, with one particularly large area in the left eye. The elevation of the discs had disappeared.

On November 15, 1955, the blood pressure was

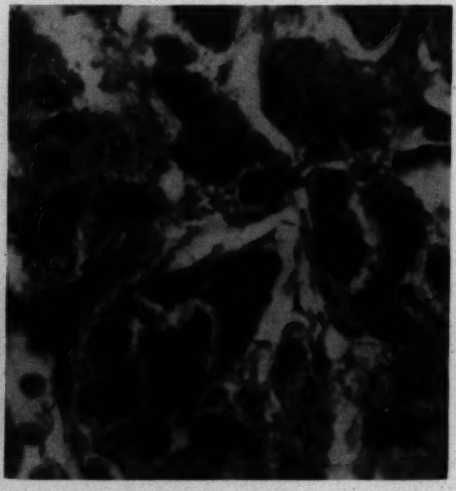

Fig. 7 (Gaines). Photomicrograph of tumor (high power).

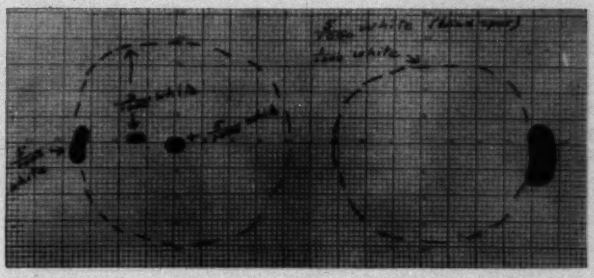

Fig. 8 (Gaines). Postoperative visual fields 11 months after operation. Compare with Figure 3.

about at the level it had been since the preceding March (124/78 mm. Hg). Vision with correction was 20/40 in the right eye and 20/200 in the left. On the near vision chart designed by Dr. James E. Lebensohn vision in the right eye was seven-point print and in the left 10-point print. The visual fields (fig. 8) showed an enlarged blindspot in the right eye. The 1/1,000 white isopter was slightly constricted. Although no central scotoma could be found, one was probably present and accounted for the decrease in vision. In the left eye, the 2/1,000 white isopter was constricted. There was a dense central scotoma for the 5/1,000 white and a paracentral scotoma for the 2/1,000 white isopter.

Funduscopic examination at this time (figs. 9 and 10) showed slight pallor of the right optic disc. The retinal vessels were normal in their size ratio. Some hard exudates were scattered throughout the retina, especially in the macular and paramacular areas. The findings in the left eye were essentially the same except that on this side there were some large, round, elevated white exudates in the macular area.

At the last examination, July 2, 1956, visual acuity was 20/50 in the right eye and 20/70 in the left, with correction. The findings were essentially the same as at the November, 1955, examination and it was assumed that the condition had become stabilized. The child had kept up with his class and passed all his school work without difficulty.

Collected cases

A search of the records of the Eye, Ear, Nose, and Throat Hospital, New Orleans, for the seven-year period ending in December, 1956, has disclosed no instance of pheochromocytoma. This would be expected; a patient with this disease requires the type of examination and treatment not provided at a specialty hospital, and any admission for the condition would be accidental.

The records of the Flint Goodridge Hospital, a local Negro institution, show only one case of pheochromocytoma over the same seven-year period. The patient, a 41-year-old

Fig. 9 (Gaines). Left fundus 11 months after operation. Compare with Figure 1.

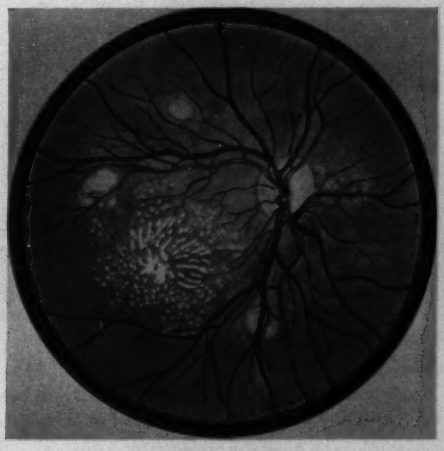

Fig. 10 (Gaines). Right fundus 11 months after operation. Compare with Figure 2.

woman, was a known hypertensive (200/140 mm. Hg) and was admitted in congestive failure six months after colostomy for carcinoma of the rectum. She died too promptly for detailed observation, but the record of an earlier admission stated that the eyes were negative; the fundi had not been examined. The pheochromocytoma was an incidental finding at autopsy. It is doubtful that the neoplasm played any significant part in the patient's symptoms or in her death, in view of the extension of the rectal carcinoma and the multiple ulcerations found in the trachea, esophagus, and intestinal tract.

Over this same period, during which total admissions numbered more than 300,000, only seven cases of pheochromocytoma were recorded at Charity Hospital of Louisiana at New Orleans, as follows:

CASE 1

A 24-year-old Negress was first seen in December, 1948, three weeks after she had been delivered by a country midwife of a healthy baby. Attacks of headaches and convulsions ensued shortly afterward, and when the patient was finally hospitalized, her blood pressure ranged from 168/120 to 206/118 mm. Hg. Pheochromocytoma was considered a diagnostic possibility but she left the hospital, against advice, before it could be confirmed.

On this admission the patient told a story of poor visual acuity for some time. Examination revealed a slight arteriovenous nicking. The outline of the disc was clear. There was questionable papilledema. Funduscopic examination was negative.

The patient was admitted for the second time August 22, 1950, with the diagnosis of toxemia of pregnancy. The blood pressure was 250/160 mm. Hg and ocular findings were essentially the same as those just described. Retroperitoneal air injection showed a mass in the superior pole of the left kidney. After removal of the pheochromocytoma on September 21st, the blood pressure fell to 110/80 mm. Hg. It was 120/88 mm. Hg at delivery October 23rd. When the patient was readmitted for cholecystectomy in April, 1956, the blood pressure was 110/80 mm. Hg and the fundi were normal.

CASE 2

A 19-year-old Negro was admitted in December, 1952, with a history of abdominal cramps, dizziness, vomiting, and blurring of vision for the past five months. If he read during the periods of blurred vision, he suffered frontal headaches. Occasionally he saw red flashes. No abnormalities had been detected at his physical examination for service in May, 1952, and he was classified as 1A, though he had not yet been called to duty. He had consulted an "eye doctor" (apparently an optometrist) before admission and had been told that his eyes were "all right" but that he had high blood pressure.

The blood pressure on admission was 180/130 mm. Hg; four days later it was 230/185 mm. Hg. The benodaine (Piperidylmethyl benzodioxane) test was positive. The discs were slightly hazy at the periphery and there was physiologic cupping (three diopters). There was a universal retinal exudate. The arteriovenous ratio was 1:3 and nicking was present. No hemorrhages were observed.

A pheochromocytoma of the left hypogastric fossa was removed April 7, 1953. Recovery was uncomplicated. Visual acuity May 5th was 20/200 in the right eye and 20/30 in the left. The intraocular pressure was 17 mm. Hg (Schiøtz).

CASE 3

A 56-year-old Negro was admitted January 24, 1954, with a history of nausea, headaches, and nocturia for the past several years. The blood pressure on admission was 210/110 mm. Hg. Renal angiograms and roentgenologic examination after perirenal air insufflation revealed a 12 by 17 cm. mass in the right adrenal region. A pheochromocytoma was removed in April, 1954. Recovery was uncomplicated. A letter from the patient a year later stated that he was perfectly well and relieved of all symptoms. There is no mention of the eyes anywhere in this record.

CASE 4

A 62-year-old Negro died January 27, 1955, 13 days after he had been admitted hemiplegic and in shock. He had a long history of hypertensive cardiovascular disease, with a blood pressure ranging from 150/90 to 180/104 mm. Hg. Autopsy showed that he had died of a massive cerebral hemorrhage and multiple pulmonary emboli. It also revealed a pheochromocytoma of the right adrenal gland one by one mm. in extent.

On admission, the patient's eyes were fixed in right lateral gaze and the pupils were miotic (one mm.). Funduscopic examination was unsatisfactory, but neither hemorrhage nor exudate were observed. There was some arteriolar spasm and some arteriovenous nicking. The only previous mention on the record of the eyes was a statement seven years earlier that the fundi were negative.

CASE 5

A 31-year-old Negress was admitted January 4, 1955, with a story of headaches for six months. It was specifically stated that she had no complaints referable to the eyes. The blood pressure, which

was 270/170 mm. Hg on admission, rose to 300/190 mm. Hg on the Benodaine test. Two tests with Regitine both gave equivocal responses. A number of examinations revealed grade II retinopathy (spasm and arteriovenous nicking). The arteriovenous ratio was 4:1.

CASE 6

A 17-year-old Negress was admitted in August, 1955, with a fibro-adenoma of the breast. Her blood pressure was 120/100 mm. Hg. She stated that she had been operated on elsewhere, in 1952, for pheochromocytoma, and had experienced cardiac arrest during an episode of extreme hypertension. There is no mention of the eyes on the hospital record. The patient's story, though it lacked confirmation, was considered credible.

CASE 7

A 29-year-old Negress had a long history of treatment in the hospital and clinic for "schizophrenic" symptoms, as well as cardiac disease. In 1947, after her seventh pregnancy, she was told that her blood pressure was 200/120 mm. Hg, and her tubes were tied bilaterally, in an endeavor to prevent the development of malignant hypertension. Between that time and her hospitalization in January, 1956, she was followed in the clinic. Her blood pressure ranged from 140/90 to 210/120 mm. Hg, and electrocardiograms showed signs of myocardial disease. When a Regitine test was performed in March, 1955, the pressure fell from 160/140 to 135/80 mm. Hg.

The patient was hospitalized in January, 1956, complaining of headaches, dizziness, and staggering gait. Visual acuity was good, but arteriolar spasm and arteriovenous nicking were demonstrated in the left eye. The blood pressure was 280/150 mm. Hg on admission. When the Regitine test was repeated, it fell from 215/120 to 170/90 mm. Hg within one minute. The electrocardiogram at this time showed no definite abnormalities. Roentgenologic examination (KUB) gave the impression of a soft tissue mass in the left suprarenal region.

In September, 1956, the patient's condition was about the same. Vision was 20/30 in the right eye and 20/50—2 in the left. The fundi were poorly seen but no papilledema was evident.

Although the diagnosis in this case is unquestionably pheochromocytoma, the patient has not yet accepted operation. She is receiving Regitine therapy.

In four of these seven cases from the New Orleans Charity Hospital, the diagnosis of pheochromocytoma was confirmed by operation or post-mortem examination. In another case, the patient's story of previous surgery for this condition seems highly credible. In at least one of the two nonsurgical cases, the diagnosis is unquestionable and in the other it is highly probable.

GENERAL CONSIDERATIONS

Pheochromocytoma is for all practical purposes a new disease; it has become a condition of clinical importance only within the last 30 years.[1] It is now realized, however, that Fraenkel, in 1886, described the condition clinically; in the light of today's knowledge, there is no doubt that the bilateral tumors found at autopsy on his patient were pheochromocytoma. The first correct clinical diagnosis was made by Vaquez and Donzelot, in 1926, and was confirmed at autopsy. During one paroxysmal episode, incidentally, their patient had an intraocular hemorrhage and lost the sight of one eye. The first removal of a pheochromocytoma is usually credited to Mayo, in 1927. It was accidental; the patient was undergoing surgery on the sympathetic nervous system for presumed malignant hypertension, and the tumor removed from the adrenal gland was diagnosed in the laboratory as a retroperitoneal malignant blastoma, its true nature not being realized until later. In 1929, Pincoffs[5] made the first correct preoperative diagnosis; the tumor was removed by Shipley and the patient made a good recovery.

Pheochromocytoma is still an uncommon disease but it can no longer be considered rare. Several hundred cases have been recorded, and hundreds of others have undoubtedly been observed though they have not been reported. Diagnostic refinements, particularly the introduction of Regitine, have made its recognition relatively simple if the physician's index of suspicion is high enough. In the 15 cases reported by Minno and his associates,[6] which were observed at the Mayo Clinic in 15,984 consecutive post-mortem examinations between 1928 and 1951, pheochromocytoma was mentioned as a diagnostic possibility in only three of the 13 cases in which the patients were fit for investigation. More typical of the present

status of the disease is the report by Grimson and his group[7] of five cases, all diagnosed correctly, all treated surgically, and all observed within a two-year period.

The small number of cases, seven, observed at the New Orleans Charity Hospital over a seven-year period in which there were more than 300,000 hospital admissions raises the question whether these figures can be accepted as absolute. It is possible that other patients with this condition were considered to have other diseases, particularly the toxemia of pregnancy, as will be pointed out shortly, or long-sustained hypertensive vascular disease. A more liberal use of the Regitine test, as has already been mentioned, might have brought other cases of the latter condition to light.

Cahill's[8] report, in 1953, from the Presbyterian Hospital in New York, demonstrates conclusively the increasingly hopeful outlook in pheochromocytoma. Seventeen cases were observed between 1924 and 1952. The only two patients admitted between 1924 and 1939 died after surgery, which was, however, undertaken to relieve their symptoms, not to correct the primary disease. One of the 13 patients admitted between 1941 and 1952 was not operated on, but surgery was done in the other 12 cases with no deaths and with relief of symptoms in every instance. Such a report indicates not only the increasing recognition of pheochromocytoma but also the increasing safety of modern surgery.

As many writers have pointed out, the selection of the anesthetic is important and so are the details of surgical technique; however, what matters most of all is a complete understanding of the physiologic principles upon which the management of these cases is based. It is a revelation, in fact, to read the accounts of the elaborate and almost dramatic routines employed in the operating room to ensure the patient's safety during the removal of a pheochromocytoma.

Review of Literature

A review of the literature of pheochromocytoma from the ophthalmologic standpoint is both revealing and frustrating. Rodin,[2] in 1945, and Bruce[3] and Hollenhorst,[4] in 1948, were apparently the first observers to make special reports on this phase of the disease. They are apparently, also, the only observers to interest themselves primarily in ocular involvement. Rodin's and Bruce's articles appeared in the *Archives of Ophthalmology*. Since then, contributions have appeared in a remarkably wide range of medical journals, including those of general interest and those devoted to internal medicine, surgery, gastroenterology, pediatrics, urology, and endocrinology, among other specialties. In DeCourcy and DeCourcy's[1] text, however, no article is listed in their extensive bibliography in any other journal of ophthalmology, nor have I found such a contribution in the five-year review of the literature with which I have supplemented their study.

Among the difficulties attending this five-year review of the literature of pheochromocytoma is one which Bruce[3] pointed out in 1948, the very considerable duplication of reports of the same case. Some cases found in my own review were recorded, by different authors, or different groups of authors, or by the same authors in different journals, sometimes as many as four or five times. Without alertness and a good memory, the reader could easily use the same case several times in any collected series.

Most frustrating of all, to one reading the literature from the standpoint of ophthalmologic involvement in pheochromocytoma, is the inadequate reporting of these important data. In the seven cases reported from the New Orleans Charity Hospital, one cannot fail to be impressed by the casual reporting of the state of the eyes or the absence of any statements at all about them. Even in the cases in which the descriptions are most generous, there is still not a great deal of information, and in two cases (Cases 3 and 6), the eyes were not mentioned in either the history or the physical examination. The rec-

ords at this hospital are typical of the case reports in the literature. In a surprising number of articles, either the descriptions are so slight as to be of little use or the eyes are not mentioned at all.

For this state of affairs there are a number of reasons: Some patients were so near death when they were observed that no elaborate diagnositic routine was possible. Some of the articles were written for a special purpose, to describe modifications of the surgical technique, for instance, or some special diagnostic routine, and the authors were not concerned with other matters. Furthermore, as Bruce[8] points out, the omission of details about the eyes does not necessarily mean that the eyes were not studied. All of this is true, but it is no help to the reader seeking ophthalmologic information. Furthermore, as Bruce also points out, some of the descriptions, particularly the negative descriptions, perhaps should not be accepted at their face value. It is not always clear whether statements that the fundus was normal were based on observations by an internist who knew how to use the ophthalmoscope or by an experienced ophthalmologist, who might be expected to see details with this instrument which a physician not thoroughly trained in the specialty might overlook.

Another regrettable fact revealed in a review of the literature is that in a surprising number of cases in which ophthalmologic changes are described before operation, there is no record of the postoperative findings. Regression of serious ocular changes can follow operation, as my personally observed case shows and as some recorded cases also show, and it is unfortunate that so few cases in the literature make any mention of that very hopeful possibility.

Finally, authors who described ocular changes in their cases, sometimes in a fair amount of detail, often failed to list them, let alone emphasize them, in their discussion of symptoms and signs. This was true of both personally observed and collected series. Smithwick and his associates,[9] who analyzed 107 collected cases and reported 11 which they had personally observed, mentioned constriction of the retinal arteries in their clinical discussion but failed to list it (or any other ocular symptom or sign) in their otherwise excellent tabulated list of diagnostic criteria, except for its inferential inclusion among peripheral vasomotor phenomena.

Comparison of the ocular changes of pheochromocytoma and optic neuritis, as already mentioned, does not seem to be recorded anywhere in the literature. DeCourcy and DeCourcy[1] in their excellent text, which is significantly entitled, "Pheochromocytoma and the general practitioner," and in which they emphasize the tendency of this disease to mimic other diseases, do not include optic neuritis in their section on changes in the ocular fundi. They are quite correct, however, in their statement that virtually any variety of ocular change, or no changes at all, may occur in patients with pheochromocytoma. They are also correct in their emphasis on the reversible nature of these changes after operation, especially in early operation and in patients in the lower age groups.

Analysis of recently recorded cases

DeCourcy and DeCourcy's[1] text was published in 1951, and the 361 articles listed in the bibliography cover the literature through 1950. A search of the literature for the 1951-1955 period has produced about 180 articles on pheochromocytoma. About 20 of them, for various reasons, could not be examined. The remaining 160 were carefully analyzed from the ophthalmologic standpoint and all data relative to the eyes were tabulated.

Recorded series of cases

In six series of personally studied cases, ranging in number from five to 25, a total of 162 cases were recorded. Eye changes were not mentioned in 102 of these. In the remaining 60 cases, the pathologic findings included

arteriolar narrowing (53 cases), hemorrhages (38 cases), exudates (35 cases), changes at the arteriovenous crossings (17 cases), and elevated discs (13 cases). It is in the highest degree unlikely that these findings are comprehensive.

In two collected series of cases in young children, as the dates of publication (1953 and 1954) indicate, there is considerable overlapping, and the age limits, which are different, add to the confusion. Goldfarb[10] mentioned visual disturbances in four of 15 children under 13 years of age but gave no other details. Daeschner and his associates,[11] whose series consists of 16 cases in children under 14 years of age, stated that there were complaints of visual disturbances in only two cases but that retinopathy was present in all 16 and was bilateral in eight.

A similar disparity between the slightness or complete absence of visual complaints and the serious ophthalmologic changes found on examination is a striking feature in many reported cases. It was also observed in the Charity Hospital series. In Cases 1 and 2, the findings suggested that the patients' complaints should be far more serious than they were, while in Case 5, in which marked pathologic changes were found, it was specifically stated that the patient had no complaints referable to the eyes.

Case reports

The 160 articles on pheochromocytoma analyzed from the recent literature include a total of 237 separate case reports, varying from one in most articles to four or five in a few instances. In 145 of these case histories the eyes were not mentioned at all. In 13 it was stated that the fundi were negative.

In 79 cases the eye changes were described in more or less (chiefly less) detail.

In these 79 cases, there were 47 males. Their age distribution in 45 stated cases ranged from four to 57 years of age (to nine years, five; to 19 years, five; to 29 years, eight; to 39 years, 14; to 49 years, 10; to 57 years, three).

There were 32 females, whose age distribution ranged from eight to 66 years (to nine years, one; to 19 years, three; to 29 years, eight; to 39 years, seven; to 49 years, seven; to 59 years, four; to 66 years, two).

It would take a much larger series of cases to determine whether these age groups are of any significance, and, particularly, whether the greater number of older patients in the female group represents a chance distribution, as is most likely, or indicates a trend. For the ophthalmologist, the important fact is that pheochromocytoma is a disease which may occur at any age, including the extremes of life, and that it will most certainly be overlooked if any attempt is made to exclude it on the basis of either age or sex.

The data concerning race are of little value in this analysis of the literature, for the point was not usually mentioned. The disease was reported in Batu, Ceylonese, Egyptian, Hawaiian, and Mexican patients, and in at least four Negroes. The fact that all seven patients at the New Orleans Charity Hospital were Negroes is interesting, in view of the small number of patients specifically described as Negroes in reported cases and series. The incidence, however, can scarcely be regarded as significant, since admissions are now running about 70 percent Negro, and since seven cases represent too small a number, in any event, to warrant generalizations.

Visual complaints

In 39 of these 79 cases, subjective complaints either did not exist or were not recorded, though, in all, changes were found when the eyes were examined. Nine of the patients were children, ranging in age from four to 12 years, and visual impairment of minor degree might perhaps be overlooked at this period of life. In one case reported by Iseri and his associates[12] in an eight-year-old Negro child, the first indication of failing vision was poor school work. In my own case, it was interference with his school work which finally brought the child to an ophthalmologist.

Subjective complaints in these cases ranged

from transient blurring of the sight to actual blindness. One patient was described as suffering from episodic blindness, but the description of the eyes, unfortunately, did not mention the state of the fundus.

PATHOLOGIC FINDINGS

Although ocular changes had been mentioned in earlier case reports of pheochromocytoma, Rodin,[2] in 1945, was the first to describe them in any detail. His patient presented extensive, progressive changes in the retina, characterized by vascular involvement, hemorrhage, and exudation. The discs were not affected, and the changes regressed after operation. The article is well illustrated.

In 1948, Bruce,[3] in an extensive article dealing with the whole subject of pheochromocytoma, reported three cases, all in children, in all of whom retinal changes were present. Papilledema was also present in one case. One patient died on the operating table. Another had normal vision and normal fundi five months after operation. The third patient presented entirely normal findings in one eye and considerable improvement in the other shortly after operation. Hollenhorst[4] also described changes in the eyegrounds, with very considerable improvement 18 months after operation.

Because of the variety of descriptions and the widely varied nomenclature employed in the 79 cases in the recent literature in which ocular findings are listed, it is extremely difficult to reduce the data to a common denominator. The following tabulation is presented, with the full realization that it is superficial and unsatisfactory:

Arteriolar narrowing was listed in 50 cases. In one instance the vessels were described as tortuous, and in another the involvement was stated to be both focal and general.

Hemorrhages were present in 33 cases. In two instances they were described as old, in one as single, in one as large, in one as subretinal, and in one as both subarachnoid and subretinal.

Exudates were present in 28 cases. In one instance they were described as old.

Elevation of the disc (papilledema) was present in 31 cases. In two instances it was described as slight.

Changes at the arteriovenous crossings were described in 22 cases.

Rodin[2] correctly states, in the first published article devoted solely to ocular changes in pheochromocytoma, that serial studies are necessary to give the complete picture. A number of reports in the literature bear out this observation and make clear also the harmful possibilities inherent in this neoplasm as far as both eyes and life are concerned.

Crede and Kerr,[13] for instance, reported a 43-year-old patient with paroxysmal episodes caused by pheochromocytoma. Presumably he had no subjective visual complaints and no fundal changes when he was first seen. Three years later, he presented moderate generalized narrowing of the retinal arterioles, with localized areas of more pronounced narrowing. There was no statement concerning his eyes after operation, but it can be assumed that the changes regressed, since the condition was mild enough to be reversible.

Stokes,[14] in the best serial description encountered, reported the case of a 28-year-old man with persistent hypertension whose eyesight was good and whose visual acuity was normal when he was first seen but who had bilateral papilledema on examination. Three weeks later striate hemorrhages and patches of exudate were present bilaterally. Five months later there was a bilateral disc elevation measuring four diopters. Five weeks later a large mass of exudate was observed near the lateral aspect of the right optic disc. Three months later there were large masses of exudate and star-shaped figures in the macular regions in both eyes. Death occurred one month later, after rapid general deterioration. Four months earlier the patient had refused sympathectomy, which was advised because of persistent hypertension, and bilateral malignant pheochromocytoma was an unexpected autopsy finding. The startling rapidity with which

the ocular changes progressed in this case over a period of 11 months needs no comment.

Hubble's[15] patient, an 11 and one-half year-old boy, had a five-year history of abdominal pain, sweating, vomiting, and blurred vision, sometimes associated with pain in the eyes and photophobia. Vision at the first examination was 6/60 in both eyes. Massive exudates covered the optic discs bilaterally and there were some old fibrotic scars in the periphery but there was no evidence of vascular disease. One year later the exudates had disappeared, leaving scarred choroids and retinas. Three years later the scars of the choroid were still present and there was extreme arteriolar narrowing bilaterally. The discs were not choked. The blood pressure was in the range 150/100 to 240/130 mm. Hg.

The patient died seven hours after an exploration that revealed a pheochromocytoma in the right adrenal gland and the tail of the pancreas. Autopsy disclosed four tumors, one in each adrenal gland and two accessory tumors, one on either side of the aorta.

The exudates seen at the first examination, which later regressed, were perhaps a retinopathic expression of hypertension present in a paroxysmal phase of the tumor. A number of observations in the literature, including those by Kvale and his associates,[16] suggest that patients with paroxysmal hypertension do not show fundal changes consistently until the disease has been present over a period of years; those who have had attacks only paroxysmally and over a period of months are likely to exhibit normal fundi between attacks.

One is impressed in some of the reported cases, as in some of the Charity Hospital cases, by the long intervals between ocular examinations. In Case 4 from this hospital, for instance, it was necessary to go back seven years to secure a record of the fundi. As the cases just described make clear, a great deal may happen in the eyes of a patient with pheochromocytoma over considerably shorter periods of time.

Ocular involvement in pheochromocytoma associated with pregnancy

The association of pheochromocytoma and pregnancy was observed in one of the cases reported from the New Orleans Charity Hospital (Case 1) and in 11 of the individual case reports in the literature. In seven of these 12 cases (including the Charity Hospital case) the attacks began during pregnancy, in three the symptoms appeared after delivery, and in two they had been present before gestation.

There were two deaths in the 11 cases of pheochromocytoma in pregnancy reported in the literature between 1951 and 1955. One patient underwent section at seven months for presumed toxemia, for which, incidentally, most of these patients were treated.[17] A month later her blood pressure was 250/140 mm. Hg and her vision was seriously impaired. Examination revealed bilateral papilledema, retinal hemorrhages, and narrowing of the retinal arteries. She died of a cerebral hemorrhage four days after bilateral splanchnic section. Meantime, there had been further impairment of her vision.

In the other fatal case, reported by Peelen and De Groat,[18] the patient's vision had become progressively impaired during her pregnancy and she was almost blind when she was hospitalized. Examination of the eyes revealed papilledema and retinal hemorrhages (grade IV). The consulting ophthalmologist recommended prompt cervical section, in an endeavor to save her sight. Death occurred shortly after operation. When the temperature reached 105°F., the diagnosis of eclampsia was abandoned, but, as in the other fatal case, pheochromocytoma was not considered until autopsy revealed it.

Peelen and De Groat collected from the literature 29 other pregnancies associated with this neoplasm in 19 patients. Eight women died promptly and there were two deferred deaths, both within a month after delivery.

Disturbances of vision were present in 10 of the 30 pregnancies (including their own case). They were the most prominent find-

ing in several instances but were not mentioned in the other 20 cases. Curiously, in spite of the impressive changes in their own case, Peelen and De Groat do not list ocular changes in their tabulation of diagnostic symptoms and signs. The lack of emphasis is unfortunate, for these changes are always important in the toxemia of pregnancy, and in some instances are the first indication of trouble.

The ocular changes were serious in most of the cases in which they were recorded, but they had regressed in the only two in which there is a statement concerning postoperative results; in one of these cases, postoperative observation covered a period of four years. In the case of pheochromocytoma associated with pregnancy in the Charity Hospital series, the eye changes also regressed after operation and there was no residual evidence of them at the end of five and one-half years.

Postoperative observations

Rodin,[2] Bruce,[3] and Hollenhorst,[4] in the first (and only) detailed discussions of ocular changes in pheochromocytoma, all demonstrated that these changes are reversible if the diagnosis is made promptly and treatment is instituted without delay. My own case and the surgical cases treated at the New Orleans Charity Hospital confirm that observation, as do cases in the literature recorded by Iseri and his associates,[12] Hamilton and his associates,[19] Roth and her associates,[20] Lewis and Knight,[21] Hoffman and Longmire,[22] and Popper and Theron,[23] among others. In the cases reported by Hoffman and Longmire, Popper and Theron, and Roth and Iseri and their groups, the changes were extremely serious, but in all four cases, as in my own, they had markedly regressed or entirely disappeared at the last recorded observation.

In 31 of the 43 surgical cases in which the patients survived surgery the eyes were not mentioned after operation. In another case, in which the fundi were not described before operation, it was stated that they were normal afterward. In some of these 43 cases there was also no statement concerning symptoms or signs in the preoperative histories. In other cases, symptoms included blurring of vision, spots before the eyes, acute deterioration of vision, and even episodic blindness. The findings included pallor of the discs, papilledema, hemorrhage, exudation, and changes in the retinal arteries. It is unfortunate that the postoperative results are not stated.

In all five cases reported by Grimson and his group,[7] the postoperative results were good. Before operation three patients had retinal abnormalities, including flame-shaped hemorrhages and focal spasm, or generalized attenuation of the arteriolar vascular bed. Three had exudation and blurring of the disc margins or early papilledema. Hemorrhages disappeared within a month after operation, but residual exudation disappeared more slowly. In one case it was no longer evident at 14 months, and in another it was represented at 15 months only by small white dots.

Comment

The diagnostic routine of pheochromocytoma is not, of course, the business of the ophthalmologist. His duty in that regard is simply to lose no time in referring any patient who might have the disease to a competent internist. He should, however, be aware of the clinical basis, in addition to eye changes, on which the neoplasm should be suspected.

The clinical onset of pheochromocytoma may be insidious, as in the case which I am reporting. The disease may assume a mild chronic, or a violent paroxysmal, form, as is evident in many of the reported cases. It may manifest itself under a variety of symptoms, ranging from such ordinary ones as headaches to such unexpected ones as projectile vomiting and Raynaud's syndrome. The list is long, the clinical picture is varied and bizarre, and some of the case histories are almost frightening.

The clinical picture may suggest, in addition to malignant hypertension, myocardial

disease, diabetes, psychoneurosis, thyroid disease, and renal disease. It is not surprising that some cases are mistaken for toxic thyroid disease and that thyroidectomy is done. It is surprising, in fact, as some observers have said, that this does not happen more often. As for renal disease, as Schleisner[24] emphasizes, the lack of correlation between the extreme hypertension and the mild renal manifestations should be enough to put the physician on his guard.

Pheochromocytoma should be thought of as a possible diagnosis in any young, healthy individual who goes into sudden shock or apparent cardiac failure after some apparently trivial strain, trauma, or surgery. One wonders whether its presence may not underlie some of the unexplained deaths which occur suddenly in young men in service after exertion which their comrades have endured without difficulty. Incidentally, in a surprising number of cases reported in the recent literature there was a story of hypertension recognized in service, or noted on discharge, for which no investigation was conducted.

Some observers believe that the diagnosis is easier to make in children because there is less to exclude. Although the point is not brought out in the literature, this is certainly true of the ophthalmologic involvement. Cahill[8] considers that children are more likely to have multiple tumors than adults, but this consideration is of no diagnostic aid; there is no apparent connection, ophthalmologically or otherwise, between the pathologic manifestations and end-results and the duration of the disease, the severity of other symptoms, or the total tumor tissue. In the familial group of three siblings reported by Roth and her associates,[20] the patient with fulminating hypertension and severe retinopathy, who was the most seriously ill, did not have the greatest total volume of tumor tissue.

Smithwick and his associates[9] outlined an excellent diagnostic routine (unfortunately omitting examination of the eyes), with emphasis on observations and tests which can be made by any competent physician without resort to special or unusual diagnostic facilities. Since the introduction of Regitine, a confirmatory test has been available which has the special advantage, in addition to its accuracy, that it is not associated with the risks inherent in some of the earlier tests used to confirm the diagnosis by precipitation of an elevation of the blood pressure. More than one disaster,[25] including the loss of sight,[26] has been reported after the use of these agents.

When the Regitine test is positive, no time should be lost in performing a retroperitoneal air injection to visualize the mass, and, if it is adrenal, to determine its location and extent. Surgery should follow with equal promptness.

The ophthalmologist who encounters a case of suspected pheochromocytoma would do well to remember that time is not on the side of the patient in this disease. That has already been emphasized from the ophthalmologic standpoint. It is also sadly true from the standpoint of life.

There were 23 deaths in the 79 individual cases recorded in the recent literature and analyzed in this communication. Two deaths occurred in pregnant women. Two patients died on the operating table, and one died after operation. In the latter case, the preoperative ocular findings included numerous fresh hemorrhages and exudates, arteriolar spasm, marked arteriovenous nicking, and moderate papilledema. Nystagmus, more marked on the right, was also present. After operation it was stated that the pupils were dilated, more notably on the right, and that there was no response to light.

The remaining 19 patients died without operation, five of them suddenly. In one of these cases death occurred 48 hours, and in another 24 hours, before operation was scheduled. All five patients in this group had eye changes, most of them severe, but there was nothing in these changes or in the total clinical picture to indicate that the fatality

was impending, and many of the patients who recovered after operation had seemed just as seriously ill.

The lesson of these deaths is obvious: Once pheochromocytoma is suspected, diagnostic measures should be instituted without delay, and, once the diagnosis is made, surgery should be carried out as soon as the patient can be prepared for it.

One of these cases, reported by DeCourcy,[27] carries a useful lesson. The patient died a few hours after the tumor was removed; her attacks had begun after a minor breast operation. One of Lukenmeyer's[28] patients had three acute episodes after an appendectomy.

One cannot fail to be impressed, from these and other recorded cases, by the danger of surgery in this disease when operation is done in ignorance of its existence. There is no doubt that its unsuspected presence accounts for some of the unexplained fluctuations in blood pressure sometimes observed at operation for other conditions, as well as for some sudden deaths after operation. In five of the 15 cases reported by Minno and his associates,[6] death occurred from surgical shock after surgery not related to the tumor. Patients with pheochromocytoma, as these and other cases show, are particularly susceptible to shock after incidental surgery. The final warning for the ophthalmologist is that he should bear that fact in mind and undertake no ocular surgery for patients with hypertensive disease, even if their eyes show none of the changes characteristic of pheochromocytoma, without screening them by the Regitine test to be certain that this tumor is not present.

Summary and Conclusions

Although pheochromocytoma has been recognized as a clinical entity for not more than 30 years, it can no longer be considered a rare disease. Many hundreds of cases are on record in the literature. With the diagnostic refinements that have been introduced, particularly the Regitine test, the tumor is being recognized with ever increasing frequency. It is highly probable, however, that some—perhaps many—cases are still being diagnosed as hypertensive disease and that lives are being lost as a result. It is equally probable that some cases are also being treated in error, and are likewise being lost, on the mistaken diagnosis of toxemia of pregnancy.

Ocular changes characteristically occur in many cases of pheochromocytoma, but their significance was late in being appreciated, and shockingly little attention has been paid to them. A review of the literature of this subject covering the period 1951 to 1955, inclusive, shows that such changes are mentioned in not more than a third of the 237 case reports made during this period and that the descriptions which are included are frequently too inadequate to be of value for analysis.

The lack of emphasis upon the ocular manifestations of pheochromocytoma is particularly unfortunate because these changes, if recognized promptly enough, are usually completely reversible. If diagnosis is delayed, and the tumor is not removed, life may be lost, as well as vision.

The ocular changes observed in pheochromocytoma are most likely to be considered those of hypertensive vascular disease. When they are observed in pregnancy, they may be considered those apparent in the preeclamptic state. It is believed that their confusion with optic neuritis is being reported for the first time in this communication. As the case report shows, prompt investigation of the underlying factors led to the correct diagnosis, prompt surgery resulted in almost complete disapperance of the pathologic processes in the retina and the disc, and the child's sight was saved, as well as his life.

The varied manifestations of pheochromocytoma make it the responsibility of the general practitioners and of specialists in many fields. The ophthalmologist must assume his share of the burden of recognition, for, as the case report in this thesis shows,

he may be consulted first. Unless he maintains a continuously high index of suspicion, he may have to reproach himself with loss of life as well as loss of sight.

It is unfortunate that post-mortem examination does not routinely include an examination of the eyes. A vast amount of information concerning ocular changes in pheochromocytoma was lost by failure to examine the eyes in the fatal cases found in the recent review of the literature, in the great majority of which autopsy was secured.

An ophthalmologist is perhaps not the proper physician to take the lead, but it does not seem unreasonable to suggest that one more registry, a registry of pheochromocytoma, be added to the registries already in existence. If it is set up, it is suggested that the form used should include certain specific information in which this investigation has shown many of the cases reported in the literature to be deficient. Among these data are the nature of the hypertension (paroxysmal, persistent, or, sequentially, both); the duration of the disease (whether chronic or acute); and the condition of the eyes, with specific statements about symptoms, specific details of the findings on examination, and specific postoperative results with reference to the eyes.

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References

1. DeCourcy, J. L., and DeCourcy, C. B.: Pheochromocytoma and the General Practitioner. Cincinnati, Barclay Newman, 1952.
2. Rodin, F. H.: Hypertensive retinopathy associated with adrenal medullary tumor (pheochromocytoma): A new clinical entity. Arch. Ophth., **34**:402-407 (Nov.-Dec.) 1945.
3. Bruce, G. M.: Changes in the ocular fundus associated with pheochromocytoma of the adrenal gland: Report of three cases. Arch. Ophth., **39**:707-730 (June) 1948.
4. Hollenhorst, R. W.: The ocular changes associated with pheochromocytoma. Am. J. M. Sc., **216**:226-233 (Aug.) 1948.
5. Pincoffs, M. C.: A case of paroxysmal hypertension associated with suprarenal tumor. Tr. A. Am. Phys., **44**:295-299, 1929.
6. Minno, A. M., Bennett, W. A., and Kvale, W. F.: Pheochromocytoma: A study of 15 cases diagnosed at autopsy. New England J. Med., **251**:959-965 (Dec.) 1954.
7. Grimson, K. S., Emlet, J. R., and Hamblen, E. C.: Diagnosis and management of tumors of the adrenal gland. Ann. Surg., **134**:451-463 (Sept.) 1951.
8. Cahill, G. F.: Pheochromocytoma. Bull. New York Acad. Med., **29**:749-764 (Oct.) 1953.
9. Smithwick, R. H., Greer, W. E. R., Robertson, C. W., and Wilkins, R. W.: Pheochromocytoma: A discussion of symptoms, signs, and procedures of diagnostic value. New England J. Med., **242**:252-257 (Feb.) 1950.
10. Goldfarb, P. M.: Pheochromocytoma: Report of case in nine-year-old Negro male. J.M.A. Alabama, **23**:70-74 (Sept.) 1953.
11. Daeschner, C. W., Mayer, J. H., and Able, L. W.: Pheochromocytoma in a four-year-old child: Renal hemodynamic, pharmacologic, and radiographic studies. J. Pediat., **45**:141-152 (Aug.) 1954.
12. Iseri, L. T., Chandler, D., Myers, B. B., and Bayle, A. J.: Diagnosis of Pheochromocytoma. J. Michigan State M. Soc., **50**:1370-1377 (Dec.) 1951.
13. Crede, R. H., and Kerr, W. J.: Pheochromocytoma: Report of a case with symptoms simulating acute anxiety syndrome. Postgrad. Med., **11**:288-293 (Apr.) 1952.
14. Stokes, E. H.: Bilateral pheochromocytomata. M. J. Australia, **1**:144-146 (Feb.) 1952.
15. Hubble, D.: Pheochromocytomata in children. Arch. Dis. Childhood, **26**:340-350 (Aug.) 1951.
16. Kvale, W. F., Priestley, J. T., and Roth, G. M.: Pheochromocytoma: Clinical aspects and surgical results. Arch. Surg., **68**:769-778 (June) 1954.
17. Richards, V., and Hatch, F. N.: Surgical experiences with pheochromocytoma. Ann. Surg., **134**: 40-54 (July) 1951.
18. Peelen, J. W., and De Groat, A.: Pheochromocytoma complicated by pregnancy. Am. J. Obst. & Gynec., **69**:1054-1061 (May) 1955.
19. Hamilton, M., Litchfield, J. W., Peart, W. S., and Sowry, G. S. C.: Pheochromocytoma. Brit. Heart J., **15**:241-249 (Apr.) 1953.
20. Roth, G. M., Hightower, N. C., Jr., Barker, N. W., and Priestley, J. T.: Familial pheochromocytoma: Report on three siblings with bilateral tumors. Arch. Surg., **67**:100-109 (July) 1953.
21. Lewis, R. G., and Knight, L. W.: Report of a case of pheochromocytoma cured by surgical removal. M. J. Australia, **1**:978-981 (June) 1954.

22. Hoffman, S. H., and Longmire, W. P., Jr.: Pheochromocytoma: Report of a case with preoperative diagnosis and removal through anterior abdominal incision. California Med., **75**:40-44 (July) 1951.
23. Popper, E. N., and Theron, P.: Phechromocytoma in a Bantu child. South African M. J., **28**:286-291 (Apr.) 1954.
24. Schleisner, P.: Bilateral phechromocytoma in a child: The differential diagnosis from nephritis. Danish M. Bull., **1**:142-146 (Oct.) 1954.
25. Bierman, H. R., and Partridge, J. W.: Untoward reactions to tests for epinephrine-secreting tumors (pheochromocytoma). New England J. Med., **244**:582-586 (Apr.) 1951.
26. Kuehn, C. A., and Arthur, J. N., Jr.: Diagnosis and treatment of pheochromocytoma. J. Urol., **68**:413-422 (Aug.) 1952.
27. DeCourcy, J. L.: Pheochromocytoma. Am. J. Surg., **86**:37-44 (July) 1953.
28. Lukenmeyer, G. T.: Pheochromocytoma. J. Indiana M. A., **47**:1377-1384 (Dec.) 1954.

USE OF GENERAL ANESTHESIA AND MUSCLE RELAXANTS IN CATARACT SURGERY*

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In the three and one-third year period, from May 4, 1954, to September 19, 1957, 586 cataract extractions were performed by various ophthalmologists at the Millard Fillmore Hospital, Buffalo, New York, using a general anesthetic together with muscle relaxant drugs. During this period only seven such operations were performed by the same ophthalmologists solely with the aid of local anesthesia. Thiopental sodium with nitrous oxide was the primary anesthetic in all cases, and tubocurarine chloride supplemented by succinylcholine chloride were the muscle relaxants used.

Since curare was first employed in ophthalmic surgery by Kirby,[1] the method of its use has been extended. It was then used only in conjunction with local anesthesia, and was usually administered by a nurse.[2] In the beginning Kirby confined it to those patients who "may behave badly even after all ordinary measures have been used," the hypertonic, hyperkinetic group for whom mental relaxation is not possible without muscular relaxation. In extreme cases, such as patients who had already lost an eye, he resorted to general anesthesia.

As a further step in obtaining complete relaxation of the cataract patient, Farquharson[3] supplemented the use of curare in conjunction with local anesthesia by including the Van Lint procedure and also retrobulbar injection. Cordes and Mullen,[4] in 1951, after using curare in 85 successive cases of cataract extraction, stated that it should be used routinely in such cases. Meanwhile both the operative technique of cataract extraction and the technique of administration of the akinetic drugs were changing. In 1953, Henderson[5] described 138 successive cataract operations in which curare alone was used to obtain akinesia; no local anesthetic agents were used except topically. At first skeptical as to its value, he stated that he was now using curare in all cataract extractions except where contraindicated. The same year Barraquer Moner[6] reported using curare with local anesthesia in all intraocular surgery. He commented that after experience with over 1,500 cases he was convinced that its use had greatly advanced ophthalmic surgery.

We have been using general anesthesia combined with muscle relaxant drugs in cataract surgery since 1954. Since using this method we have abandoned the topical application of cocaine derivatives, and the use of the Van Lint technique, the facial block, retrobulbar injection, and suture of the rectus oculi superior muscle. The anesthetic

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technique varies among the different operators. However, a brief description of our technique may be of interest.

Anesthetic procedure

Premedication is given on the evening before and the morning of operation. Morphine is not used because of its inclination to produce nausea. Meperidine hydrochloride (50 to 100 mg.), a belladonna alkaloid (0.3 to 0.6 mg.), and dimenhydrinate (50 mg.) are injected one hour preoperatively, the specific doses varying with the age, weight, and general condition of the patient. With the patient breathing 100-percent oxygen by mask, sleep is gradually induced with minimal quantities of thiopental sodium. As soon as the patient is unconscious, succinylcholine chloride is also administered intravenously, in the amount necessary to produce complete relaxation of the jaw muscles and larynx (about 60 mg.). An endotracheal tube is then placed and the pharynx packed with moist flannel gauze to absorb conjunctival irrigating solutions which drain to the pharynx.

Anesthesia is maintained by six-liter flows of a 50:50 mixture of nitrous oxide-oxygen through the endotracheal tube and by intermittent injections of thiopental sodium. When the patient's respirations return, a test dose of 3.0 mg. of tubocurarine chloride is injected intravenously; if no undesirable effects are noted within five minutes, from 3.0 to 6.0 mg. more are given, followed by increments of the drug as needed during the course of the operative procedure. The minimum total dose required for complete relaxation of the eyeball was 6.0 mg., the maximum 21 mg.

In none of our patients was any evidence of histamine release, such as a profound drop in blood pressure or bronchospasm, noted. The tubocurarine chloride was administered so as to synchronize its maximal effect with the time when the surgeon was extracting the lens. Such drugs as prostigmine and endrophonium were rarely required. Postoperatively chlorpromazine hydrochloride (12.5 mg.) is given intramuscularly while the patient is still in the recovery room and repeated every four to six hours if necessary. Dimenhydrinate (50 mg.) is substituted in patients who are hypotensive or extremely slow in recovering from the anesthetic.

Effects of the drugs

After injection of tubocurarine chloride, muscular relaxation begins in two minutes. The effects on the skeletal musculature are manifested by the sequence of ptosis, followed by relaxation of the eye muscles, then the facial muscles, the neck muscles, and finally the long muscles of the back and the extremities. The eyes are in central position and motionless. The orbicularis oculi is completely relaxed and the eyes remain in the central position throughout the operation. The recovery phase is generally quiet and uneventful, with no memory of the anesthetic procedure. Residual weakness of the extrinsic eye musculature does not persist. Numerous investigations have demonstrated that tubocurarine chloride, in clinical doses, has no central depressing effect; its pharmacodynamic action is at the neuromuscular junction.

To avoid prolonged apnea, curare should not be used in cases of myasthenia gravis. Also, if the drug has a similar pharmacologic action in the human as it does in the dog, it should be used with caution in cases of allergic asthma. However, we have used it, without reduction of dosage, in such cases with no untoward effects.

The action of succinylcholine chloride on the extraocular muscles and on intraocular pressure merits some discussion. Lincoff and his associates,[7] in 1955, had demonstrated by in vitro experiments that one of the physiologic actions of succinlycholine is to increase intraocular pressure. This was subsequently confirmed by Dillon, et al.[8] They showed, by in vivo experiments, that succinylcholine chloride caused contraction and increased tension of the extraocular muscles in cats

during depolarization. Thus succinylcholine chloride, which showed promise of being the ideal short-acting relaxant, apparently had its value negated by causing a rise in intraocular pressure.

Schwartz and deRoetth[9] produced evidence to the contrary, however, and concluded that "Succinylcholine can be used in ocular surgery in single doses for tracheal intubation preoperatively (except in patients with acute narrow-angle glaucoma in remission) since any rise in ocular tension is dissipated by the time surgery is begun. It may be used with caution throughout an operation as an intravenous drip, if it is begun well before surgery starts and the patient's succinylcholine level is maintained throughout surgery (that is, kept completely paralyzed). It should never be started after actual surgery on the eyes is begun." Indeed, in our cases the routine use of succinylcholine chloride has produced no clinical evidence of a rise in intraocular pressure.

Results in present series

Of the 586 cataract extractions in this series, 69 were extracapsular and 517 intracapsular. The patients ranged in age from 16 to 91 years, the average being 63 years; 143 patients were over 70 years of age and 22 were over 80. Nearly all types of medical conditions were represented, including diabetes mellitus, leukemia, asthma, hypertension, various cardiac conditions, polycythemia vera, and psychoses. Associated ophthalmic conditions included glaucoma, retinitis pigmentosa, and coloboma of the iris and choroid. There were three patients who had lost their sight in one eye and four who had had a corneal transplant.

Complications encountered included 12 cases of prolapse of the iris, eight cases of loss of vitreous, and one case each of cardiac arrest, expulsive hemorrhage, and endophthalmitis. None of them can be attributed to the anesthetic procedure. The case of cardiac arrest occurred in the recovery room. The patient made a full recovery, and repair of a prolapsed iris was done by the same method subsequently with no further complication. The expulsive hemorrhage occurred during incision by a keratome; the eye was enucleated. The case of endophthalmitis occurred in the oldest patient, a 91-year-old individual.

Comment

After the discovery of general anesthesia early in the 19th century, it was applied to intraocular surgery with some success. Prior to that, various soporific potions to lessen pain and relax mental tension had been used. The introduction of local anesthesia later in the same century was followed by the performance of most ophthalmic surgery with the aid only of cocaine instillation, injection of procaine hydrochloride, or both. In all ophthalmic surgery, and particularly in extraocular surgery, the proper level of anesthesia materially influences the outcome of the operation. The anesthetic used has long been considered an integral part of eye surgery, playing a more vital role than in other types of surgery. Thus the return, in recent years, by many ophthalmologists to the use of general anesthesia in intraocular surgery was a rather radical departure.

Although the muscle relaxant drugs may be employed in eye surgery in conjunction with local anesthesia, we believe they are more effectively used, and with less distress to both patient and surgeon, in conjunction with general anesthesia. For example, with the aid of a general anesthetic and the muscle relaxants, it is a simple matter to reflect the corneal flap and remove particles of capsule or cortex from the surface of the vitreum. Without these aids, the vitreous humor tends to move forward and extrude. This procedure may be further facilitated by the use of fluorescing ultraviolet light to visualize cortex and capsule more distinctly.

From the age of the patients in this series, it may be concluded that the muscle relaxants may safely be administered to eye patients of all age groups. This conclusion is

concurred in by all the ophthalmologists using the muscle relaxants in this hospital.

Should all patients having cataract surgery be given curare? We believe they may be, if consultation with the cardiologist and anesthesiologist indicates its safe use. The ophthalmologists who have performed cataract surgery with the aid of general anesthesia and these muscle relaxants agree that their work was performed under ideal conditions, with no need for hurry. The patients have benefited from better results, have seldom been nauseated, have had no postoperative complaints, and have no memories of an unpleasant induction.

Mushin[10] has raised the point that technical advances in anesthesiology are so far ahead of the number of available trained people to utilize them, that there is some uneasiness that patients may be suffering harm from these complex methods when they are used by less skilled anesthetists.

Conclusions

1. When general anesthesia was first used in cataract surgery in this hospital by one of us (E. B. H.) results were immediately gratifying and others soon followed suit. The present study was made over a 40-month period to ascertain the degree of satisfaction with its use. The fact that the group using general anesthesia continues to do so almost exclusively, we believe speaks for itself.

However, we believe there is room for difference of opinion in the technique of choice for an individual operator. Deep analgesia and sedation with meperidine hydrochloride and chlorpromazine hydrochloride or dimenhydrinate may be another excellent technique provided the surgeon has absolute psychologic domination and control of the patient. There are, nevertheless, certain maneuvers that cannot be attempted with assumption of complete safety when the patient can move his body, his head, or his eyes or eyelids.

2. The long-acting muscle relaxant, tubocurarine chloride, used in conjunction with a general anesthetic (preferably thiopental sodium) and with the short-acting muscle relaxant, succinylcholine chloride, represents an advance in intraocular surgery and obviates the need for retrobulbar and Van Lint injections as well as suture of the rectus oculi superior muscle.

3. General anesthetics should be administered only by competent anesthesiologists who have familiarized themselves with the special requirements of ocular surgery.

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References

1. Kirby, D. B.: Three years' experience with general akinesia in cataract surgery. Concillium Ophth. (1950). Brit. M. A. Acta, **2**:1094-1102, 1951.

2. Clark, W. B.: Use of curare as an adjuvant in cataract surgery. Watson-Gailey Eye Foundation Quart., **1**:34, 1949.

3. Farquharson, H.: Curare with local anesthesia in cataract surgery. Am. J. Ophth., **34**:554-556 (Apr.) 1951.

4. Cordes, F. C., and Mullen, R. S.: The use of curare in cataract surgery. Am. J. Ophth., **34**:557-564 (Apr.) 1951.

5. Henderson, J. W.: Curare akinesia in cataract operations. Am. J. Ophth., **36**:781-788 (June) 1953.

6. Barraquer Moner, J. I.: The present technique and results of the use of curare in ocular surgery. Am. J. Ophth., **36**:789-793 (June) 1953.

7. Lincoff, H. A., Ellis, C. H., DeVoe, A. G., DeBeer, E. J., Impastato, D. J., Berg, S., Orkin, L., and Magda, H.: Effect of succinylcholine on intraocular pressure. Am. J. Ophth., **40**:501 (Oct.) 1955.

8. Dillon, J. B., Sabawala, P., Taylor, D. B., and Gunter, R.: Depolarizing neuromuscular blocking agents and intraocular pressure in vivo. Anesthesiology, **18**:439-442 (May-June) 1957.

9. Schwartz, H., and De Roetth, A., Jr.: Effect of succinylcholine on intraocular pressure in human beings. Anesthesiology, **19**:12-13 (Jan.-Feb.) 1958.

10. Mushin, W. W.: Advances in anesthesia: real or apparent? N. Y. State J. Med., **57**:3279-3283 (Oct.) 1957.

PERSISTENT HYPERPLASTIC VITREOUS*

Study of a complete case with a new histologic technique

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This is a report of only one case of persistent hyperplastic vitreous (also called persistent tunica vasculosa lentis). We have seen more cases of this entity. However, the present case allows for a demonstration of virtually all the known findings and it would be only confusing to add other less complete cases to this report. The diagnosis in this case had already been made at the clinical examination and the histologist, therefore, was prepared for the histologic demonstration of the expected findings. The use of special techniques revealed some important additional information about the ocular changes in persistent hyperplastic vitreous.

Case history

This is the case of a four-month-old white boy who was born on January 18, 1958. The child was a full-term baby who had no oxygen after birth. On his first day the mother noticed that the baby had "no pupil" in the left eye. The child was first examined a few days after birth and the parents were told that the baby would never see out of the left eye and that the right eye was normal. Later the child was sent to Dr. Falls of this eye clinic for a consultation. An examination revealed normal lids and normal conjunctiva, O.U. The corneal diameter was 10 mm., O.U. The intraocular pressure was 20.3 mm. Hg, O.U. The right eye exhibited a normal cornea and pupil, clear media, and a normal fundus. The left eye, however, showed extensive clouding and scarring of the cornea, especially centrally and nasally. The anterior chamber was flat. An extensive pupillary membrane was found. The pupil was semidilated and fixed. It appeared firmly attached to the pupillary membrane. Parts of the lens were clear. Vascularized and hyperplastic vitreous, as well as coxcomblike ciliary processes, were seen. The latter were distinctly atrophic and elongated.

The family history of the baby was negative. There were three siblings in good health. The general examination of the child revealed no pathologic findings—except for the blindness, O.S. The chest X-ray films were clear. An X-ray study of the orbit showed no evidence for calcium in either eye.

Enucleation of the left eye was done on May 13, 1958. The eye was fixed in ammonium bromide formalin (Cajal solution).

Histologic examination

Method

The eye was cut in two halves both of which contained parts of the cornea, anterior chamber, lens, the macular region, and the optic nerve. One half was imbedded in paraffin and cut in serial sections. These sections were stained with hematoxylin-eosin and with the periodic acid-Schiff stain. Frozen sections were made of the other half of the eye. These sections were impregnated with the silver-carbonate methods of del Rio Hortega as described by Scharenberg and Zeman.[1]

All pictures presented in the paper are unretouched photomicrographs.

Results

The macroscopic examination revealed the eye to be of normal shape and size (21 by 20 by 20 mm.). The horizontal diameter of the cornea was 10 mm. The anterior chamber was shallow (fig. 1). The lens was shrunken and appeared surrounded by a membrane of whitish tissue. This membrane was attached to the center of the posterior surface of the cornea on one side and to the peripheral retina on the other side. The ciliary body and the ora serrata could not be seen. Many delicate filaments were found to run from the posterior surface of this membrane

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Fig. 1 (Wolter and Flaherty). Macroscopic view of one half of the eye. The shallow chamber, the cataract, the retrolental membrane, and the filaments in the posterior chamber are clearly visible.

through the posterior chamber to the retina. There was no normal vitreous found besides these filaments (fig. 1).

The microscopic examination of slides stained with the different staining methods already mentioned showed a multitude of pathologic alterations in virtually all ocular tissues of this eye.

The cornea was covered by continuous epithelium and was normal in its architecture. However, it exhibited definite scarring in the areas to which the central pupillary membrane and the anterior synechias of the iris were attached. The endothelium of these areas was completely absent.

The anterior chamber angle was closed by extensive peripheral anterior synechias. However, a normally developed trabecular system with an open canal of Schlemm was seen underneath the peripheral iris. A dense pupillary membrane was found to cover the anterior center of the lens and was firmly attached to the posterior surface of the cornea.

The lens itself was shrunken and partly cataractous. Its posterior capsule was wrinkled and showed an extensive break. Macrophages were found in the fluid lens substance of the peripheral areas of the lens (figs. 2 and 3). A dense retrolental membrane was closely attached to the whole posterior lens capsule (fig. 2). The membrane was mainly composed of fibrous connective tissue and small patent blood vessels (fig. 4). The fibers of the lens zonule and the inner parts of some atrophic ciliary processes were incorporated in the membrane (figs. 2, 5, and 6). The fibers of the zonule, the ciliary processes, and the peripheral retina were pulled toward the membrane (fig. 5). In this process the ciliary processes had become very long and atrophic (fig. 6).

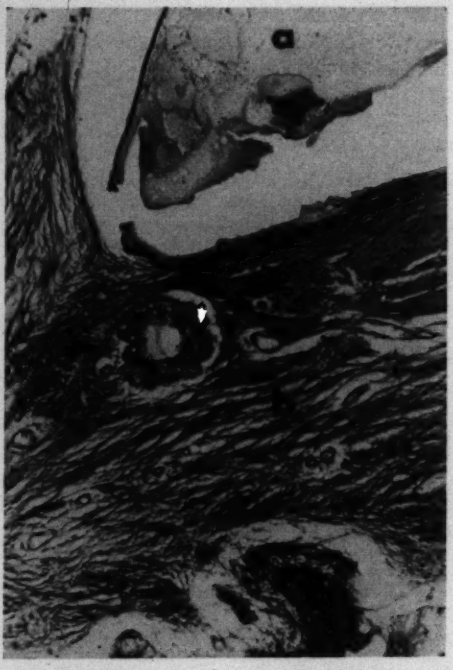

Fig. 2 (Wolter and Flaherty). Part of the cataractous lens (a) and the retrolental membrane (b). A ciliary process is seen incorporated in the membrane (arrow). The membrane also shows many small blood vessels and the peripheral retina in the lower part of the picture. (Hematoxylin-eosin, photomicrograph.)

Fig. 3 (Wolter and Flaherty). High-power view of the posterior cortex of the lens (a), a zone of macrophages in the fluid lens substance beneath the capsule, the wrinkled posterior lens capsule (c), and the retrolental membrane (b). (Hortega method, photomicrograph.)

The ciliary body was very small and atrophic and completely covered by peripheral retina. Many delicate filaments and an obliterated blood vessel were found to have their insertion in the retrolental membrane. The filaments originated all over the retina and the blood vessel came from the optic disc. The fibers of the lens zonule which connected the ciliary processes and the retrolental membrane appeared very dense and the single fibers rather thick (figs. 7 and 8).

Large areas of the peripheral retina showed extensive cystoid degeneration (fig. 9). The more central retina exhibited two different types of changes: (1) changes of the architecture of the inner retinal layers themselves and (2) changes of the inner retinal surface. The inner layers of the retina showed an extensive hyperplasia of the retinal astroglia (figs. 10, 11, and 20). The layers of the star-shaped glial cells exhibited their normal arrangement. However, their number was increased and their processes (glial fibers) formed very dense glial networks in the inner retina. In the peripheral and intermediate retina this hyperplasia of the astroglia was combined with a definite decrease in the number of ganglion cells. The ganglion cells were by no means completely missing in these areas and no degenerative changes of ganglion cells were seen. But they were just less numerous than they normally are. The central retina exhibited an about normal number of ganglion cells.

The changes of the inner retinal surface were composed of several different findings: local proliferation of glia, local patchlike accumulation of an amorphous granulated substance with or without contact to the retina (preretinal structures of Manschot[4]), and insertion of the filaments, already mentioned, which originated in the retrolental membrane.

The local proliferations of glia on the inner retinal surface were the rarest of the three changes. They represented flat patches of glial cells (fig. 12) and most of them were found in the peripheral retina. These patches of glia often exhibited different stages of hyalinization and represented in the final stage prominent hyaline bodies on the inner surface of the retina (fig. 13).

The accumulations of amorphous granulated substance were found all over the retina in all different sizes (fig. 14). It was our impression that this substance was accumulated at the inner surface of the inner limiting membrane and that it had close relations to a layer of vitreouslike substance which was found on the retina and which contained the filaments already mentioned. This layer of vitreouslike substance showed extensive shrinkage in the histologic process and broke the connections between the amorphous preretinal accumulations and the inner limiting membrane in most sections (fig. 14). The

Fig. 4 (Wolter and Flaherty). High-power view of the retrolental membrane which is composed of fibrous connective tissue (a) and blood vessels (b). (Hortega method, photomicrograph.)

amorphous substance of the preretinal accumulations contained no cells and the silver stains revealed that there was no connection between the radial fibers of Mueller or the retinal astroglia and these accumulations (figs. 21, 22, and 23). Quite often it could be observed that the filaments of the posterior chamber had their origin right on top of these preretinal accumulations (fig. 15). In some instances it was found that the amorphous substance of such accumulations was located free in the "vitreous" and had no contact with the retina (fig. 16).

The filaments of the posterior chamber which had already been seen at the macroscopic examination had their origin in the retina. The hematoxylin-eosin sections showed the filaments to be of fibrous structure and that they had close relations to the layer of vitreouslike substance on the inner

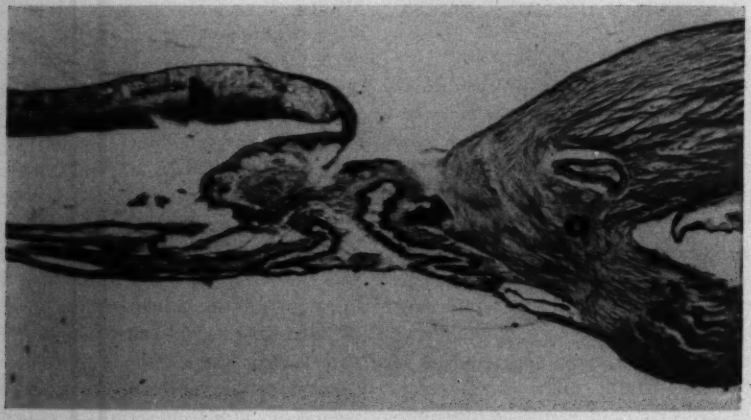

Fig. 5 (Wolter and Flaherty). The retrolental membrane (a) with the adherent retina (b). The atrophic ciliary processes can be seen attached to the membrane. (Periodic acid-Schiff, photomicrograph.)

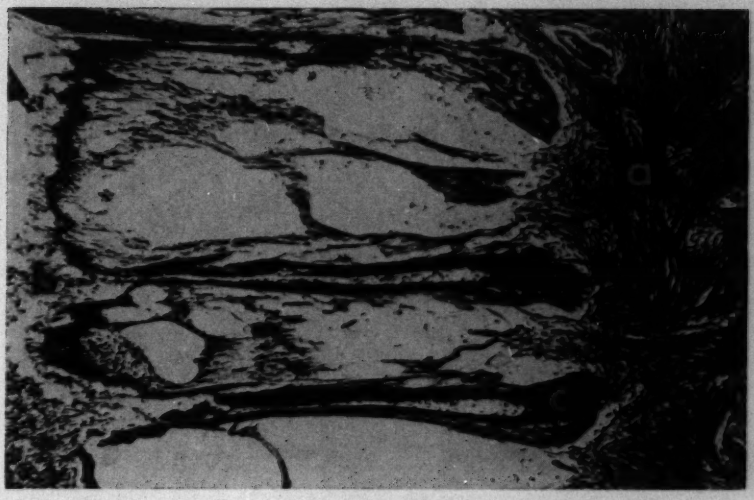

Fig. 6 (Wolter and Flaherty). Flat section through the retrolental membrane (a), the atrophic, elongated ciliary processes (c) and the ciliary body (b). (Hortega method, photomicrograph.)

retina (fig. 17) was clearly seen. Silver stains revealed that these filaments entered the retina without close relation to Mueller's radial fibers or the hyperplastic astroglia (fig. 18). It could be seen, however, that the filaments in many areas were continuous with superficial retinal blood vessels. The filaments represented dense fibrous structures without cellular nuclei and without a lumen.

The optic disc was distorted and prominent. A patent hyaloid artery was found (fig. 19). This vessel could be followed through the posterior chamber on its way to the retrolental membrane. At its origin on the optic disc it was surrounded by hyperplastic glial tissue. The optic nerve was normal in its architecture. There was, however, a distinct hyperplasia of the astroglia. The sclera was normal.

Comment

Extensive histologic studies of persistent hyperplastic primary vitreous have been pub-

Fig. 7 (Wolter and Flaherty). An area of ciliary epithelium (a) with elongated and hypertrophic zonule fibers (b). These fibers were attached to the retrolental membrane. (Hortega method, photomicrograph.)

Fig. 8 (Wolter and Flaherty). Another area of the ciliary epithelium (a) with the dense hypertrophic zonule fibers (b) in a flat section. (Hortega method, photomicrograph.)

lished recently by Sanders,[2] Reese,[3] and Manschot.[4] The reports of these authors contained all the older literature and revealed very much about the nature and etiology as well as of the characteristic pathology of this entity. The report of this single case seems worthwhile, however, since a new technique was used in it which revealed some additional histologic findings. Furthermore, it can be said that the present case is a perfect example of this entity which exhibits all the known findings of persistent hyperplastic

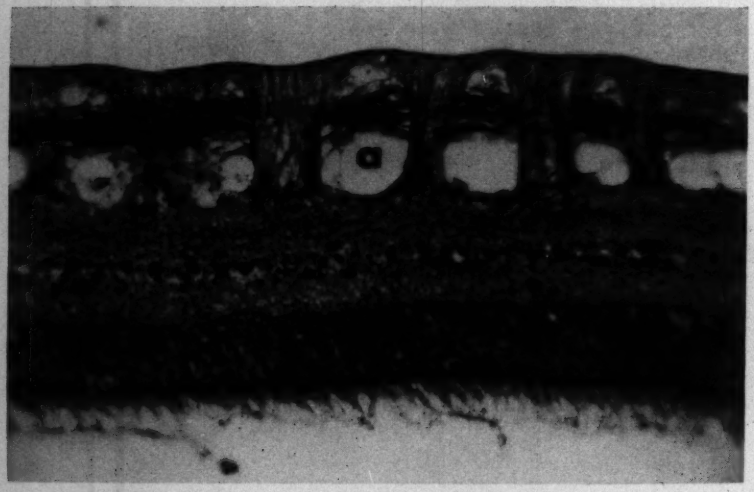

Fig. 9 (Wolter and Flaherty). Peripheral retina which exhibits extensive cystoid degeneration (a) and a complete loss of ganglion cells. (Periodic acid-Schiff, photomicrograph.)

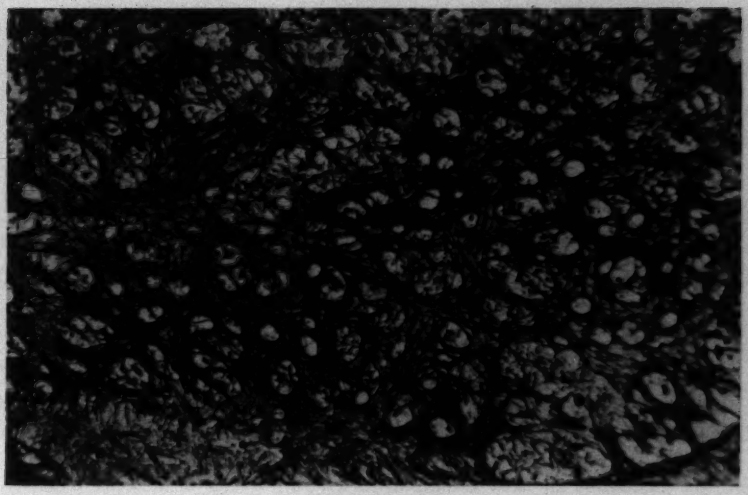

Fig. 10 (Wolter and Flaherty). Flat section through the inner layers of the central retina. The dense networks of hypertrophic astroglia are clearly visible. (Hortega method, photomicrograph.)

vitreous except for a microphthalmos and any signs of intraocular hemorrhages.

It must be emphasized that the involved eye of the present patient was of normal size and had a normal corneal diameter. Manschot[4] re-emphasized the statement of von Hippel[5] that retinoblastoma has never been found in a microphthalmic eye and that, on the other hand, microphthalmos represents one of the most important criteria of persistent hyperplastic vitreous. The present case reminds us, however, that an eye with persistent hyperplastic vitreous must not always be microphthalmic. The two other main clin-

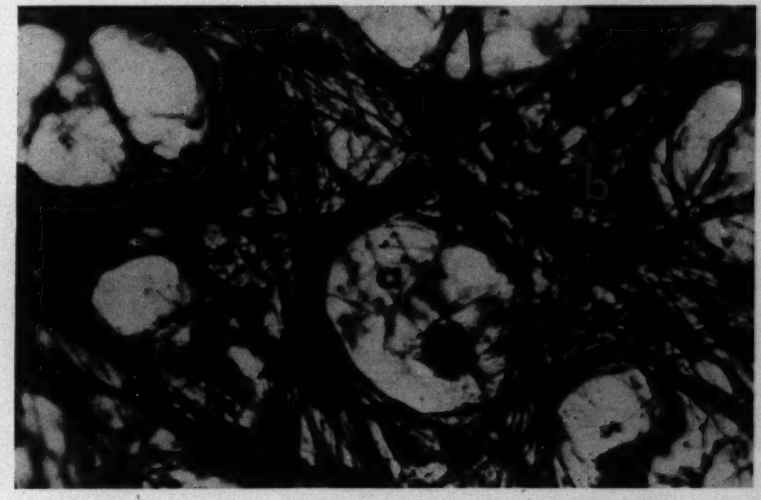

Fig. 11 (Wolter and Flaherty). High-power view of a flat section of the central retina with the hyperplastic astroglia (b) which is seen to surround ganglion cells (a). (Hortega method, photomicrograph.)

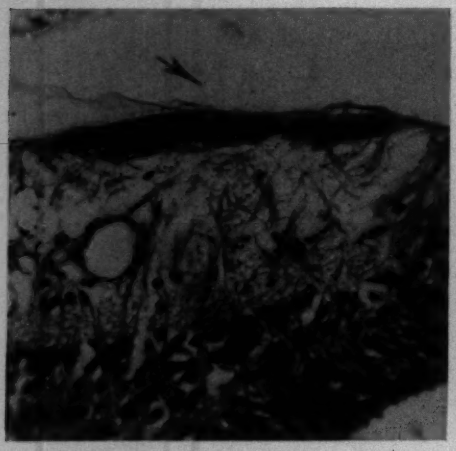

Fig. 12 (Wolter and Flaherty). Patch of proliferated astroglia (arrow) on the inner surface of the peripheral retina (Hematoxylin-eosin, photomicrograph.)

Fig. 13 (Wolter and Flaherty). Patch of proliferated astroglia on the inner retina which shows advanced hyalinization (arrow). (Hematoxylin-eosin, photomicrograph.)

ical criteria of this entity were present in this case: (1) presence of opaque tissue behind the lens and (2) elongated ciliary processes.

Histologically, this case showed all the typical findings: pupillary membrane, malformation of anterior chamber and angle, cataract with rupture of the posterior lens capsule, elongation of the ciliary processes, advancement of the peripheral retina, persistence of the hyaloid artery, fibrous filaments in the posterior chamber, lack of normal vitreous, and a multitude of pathologic changes of the inner retinal surface.

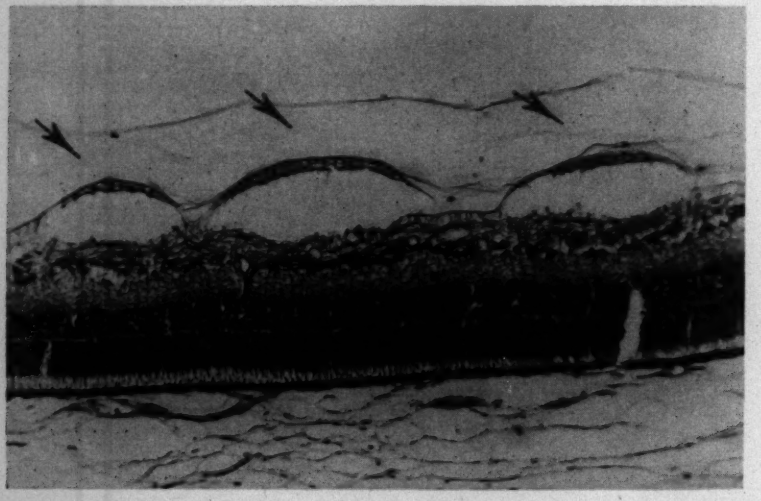

Fig. 14. (Wolter and Flaherty). Local accumulations of amorphous substance on the inner surface of the retina (arrows). These are the "preretinal structures" of Manschot. (Hematoxylin-eosin, photomicrograph.)

Fig. 15 (Wolter and Flaherty). Insertion of a small filament in a preretinal structure (arrow). The layer of "vitreouslike substance" on the inner retina is visible. (Hematoxylin-eosin, photomicrograph.)

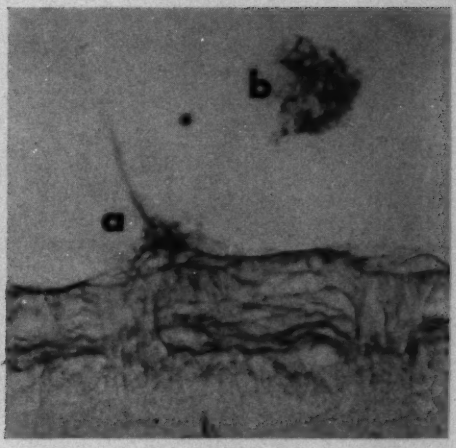

Fig. 16 (Wolter and Flaherty). Insertion of a small filament in the retina (a) and a patch of preretinal substance without contact to the retina (b). (Hematoxylin-eosin, photomicrograph.)

New findings which could be demonstrated in this case were a general hyperplasia of the retinal astroglia and a certain lack of ganglion cells in the peripheral and intermediate retina.

Reese[3] has pointed out that the fibrovascular tissue around the lens in these cases originates from the tunica vasculosa lentis. This tunica vasculosa may persist as a whole or its anterior and posterior parts may show persistence and hyperplasia independently. The present case exhibited a persistence of both parts of the tunica. The fibers of the zonule of the lens and some parts of the ciliary processes were incorporated in the

Fig. 17 (Wolter and Flaherty). Insertion of a large filament in the retina (arrow). (Hematoxylin-eosin, photomicrograph.)

Fig. 18 (Wolter and Flaherty). Silver stain of the insertion of a large filament in the retina (arrow). There is no continuation of this filament to the inner processes of the radial fibers of Mueller. (Hortega method, photomicrograph.)

Fig. 19 (Wolter and Flaherty). Optic disc (a) with the persistent hyaloid artery (arrow). (Hematoxylin-eosin, photomicrograph.)

Fig. 20 (Wolter and Flaherty). Tangential section of the retina with the hyperplastic layer of astroglia (a) and the inner processes of Mueller's radial fibers (b) which form the inner limiting membrane. (Hortega method, photomicrograph.)

posterior part of this fibrovascular membrane. And this membrane was also firmly attached to the posterior lens capsule.

Fig. 21 (Wolter and Flaherty). Low-power view of another area of a tangential section of the retina with a preretinal structure (arrow). (Hortega method, photomicrograph.)

Fig. 22 (Wolter and Flaherty). High-power view of a preretinal structure (a) on the inner surface of the retina. There is no continuation between the fibers of Mueller and the structure. (Hortega method, photomicrograph.)

It is obvious that the retrolental membrane had caused extensive traction on the fibers of the zonule of the lens, the ciliary processes, and the peripheral retina. This membrane had also caused wrinkling and rupture of the posterior lens capsule. Reese[3] explained this finding by contracture of the fibrous membrane, while Manschot[4] believed that these changes are caused by the fact that the growth of the eye exceeds the growth of the retrolental membrane. It is possible that the combination of both mechanisms may

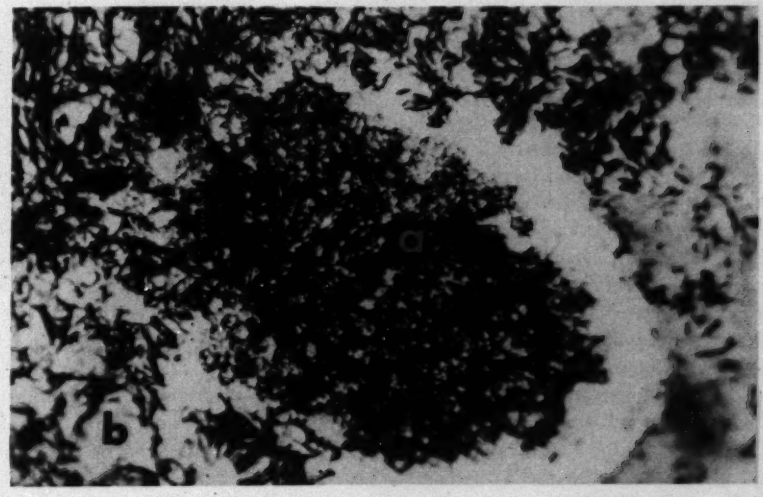

Fig. 23 (Wolter and Flaherty). Flat section of the retina with a preretinal structure (a) and the inner limiting membrane (b). These two structures show no close connections. (Hortega method, photomicrograph.)

account for the changes. The present case allowed for a demonstration of hypertrophy and elongation of the zonule fibers, atrophy and elongation of the ciliary processes, and cystoid degeneration (with loss of ganglion cells) of the peripheral retina. All these changes are considered a result of the excessive stretching of these tissues.

Malformation of the anterior chamber is a typical finding in cases of persistent hyperplastic vitreous.[2-4] In the present case the trabecular system and the canal of Schlemm were well developed. However, the latter structures were completely covered by extensive anterior synechias which bridged the anterior chamber in many areas and completely obstructed the filtration angle. It is interesting to note that the ciliary body in this area was very much distorted and covered by the advanced peripheral retina. And the ciliary processes showed extensive atrophy. The combination of these two findings may explain why the present case showed no glaucoma.

The lens also showed the typical changes of this entity:[2-4] partial cataract, wrinkling and rupture of the posterior capsule, and invasion of mesodermal elements (macrophages). The persistence of the hyaloid artery is another common finding in this condition.

Reese[3] wrote about the filaments a great number of which were observed in the posterior chamber of the present case: "Very rarely, fibrous filaments extend from the posterior surface of the retrolental tissue toward the retina and may even pull the retina away from the choroid." No normal vitreous was found in the present case and except for the filaments there was only a clear waterlike fluid in the posterior chamber. Close to the retina, however, a layer of vitreouslike substance could be demonstrated histologically. This had very close relations to the retinal ends of the filaments. Our studies with the silver methods showed that these filaments originated from superficial retinal blood vessels. This is an important finding since it supports the opinion of Reese[3] that the filaments represent degenerated blood vessels.

Manschot[4] recently demonstrated the insertion of the filaments in the retina. The same author also described the typical patches of proliferating preretinal glia (his fig. 5). Our case showed that these patches of glia may undergo hyaline degeneration. As a new finding Manschot found in some of his cases local accumulations of amorphous substance on the inner surface of the retina which he called "preretinal structures" (his fig. 7). He considered these preretinal structures "an unknown anatomic characteristic of the anomaly." We found the same preretinal structures in our case and we also found that they are often related to the insertion of the filaments in the retina. Silver stains revealed that these structures had no relation to the brushlike inner ends of the radial fibers of Mueller which form the inner limiting membrane, as well as most of the secondary vitreous. This observation supports the view of Manschot[4] that the preretinal structures are simply remnants of the embryonic primary vitreous. These may be with or without contact to the retina.

The finding of an extensive hyperplasia of the layers of astroglia in the retina is an important new observation in this case. The retinal astrocytes had their normal architecture; however, they were much more numerous than normal. Studies of the astroglia in other cases of this entity would be important to find whether this hyperplasia of astroglia represents a typical finding of persistent hyperplastic vitreous. It must be emphasized that the ganglion cells of the central retina were normal in number and arrangement despite this glial hypertrophy. In the intermediate and peripheral retina, however, there was a distinct decrease in the number of ganglion cells. This latter finding in this case is considered a secondary degeneration following the extreme stretching of the peripheral retina. The astroglia of the optic nerve also showed a certain hyperplasia of the astroglia which was less marked than that of the retina.

This paper represents an addition to the

extensive studies of Reese, Manschot, and Sanders as well as of many earlier authors. It is an instructive example for the fact that our knowledge of the details of a disease entity may be increased if new examination techniques are used.

Summary

The clinical and histologic findings of a very complete case of persistent hyperplastic vitreous in the left eye of a four-month-old child are reported. The use of a silver technique revealed interesting additional information about classic histologic changes. Furthermore, it was possible to demonstrate a general hyperplasia of the retinal astroglia which is a new finding in this entity.

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References

1. Scharenberg, K., and Zeman, W.: Zur Leistungsfaehigkeit und zur Technik der Hortega'schen Silberkarbonatmethoden. Ztschr. Psychiat., **188**:430, 1952.
2. Sanders, T. E.: Pseudiglioma. Tr. Am. Ophth. Soc., **48**:575, 1950.
3. Reese, A. B.: Persistent hyperplastic primary vitreous. Am. J. Ophth., **40**:317, 1955.
4. Manschot, W. A.: Persistent hyperplastic primary vitreous. Arch. Ophth., **59**:188, 1958.
5. von Hippel, E.: Handbuch der speziellen pathologischen Anatomie und Histologie. Berlin, Springer, 1931, v. 11, pt. 2.

TRACHOMA IN NORTHERN AUSTRALIA*

Bacteriologic and virologic aspects

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Bacteriologic flora of conjunctival and nasal mucosa of aboriginals

During the last four years various investigations into the health of the Australian aboriginal in remote parts of the continent have been undertaken both by the Public Health Departments of Western Australia and Queensland, and by the Commonwealth Department of Health. The ophthalmic investigators (Ida Mann, Western Australia; Frank Flynn, Northern Territory, in particular) have shown that the greatest single eye disease among them is trachoma. The high incidence of this disease in the remoter areas was a surprise, as the blindness rate was not high and the usual complications of trachoma were rare. The type of trachoma found was that known as "trachome pur" in which there are few or no symptoms, no discharge, and no obvious signs of disease unless one everts the upper lid and finds the typical necrotic follicles and fine conjunctival scars.

The difference in severity of trachoma in various areas of the world has usually been considered as conditioned by the presence and nature of secondary bacterial infection. The bacteriology of secondarily infected trachoma has been extensively studied in Egypt, Tunisia, and North Africa generally and this study has revealed a host of invaders, pneumococci, gonococci, streptococci, Koch-Weeks bacilli, staphylococci, and so forth,

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* From the Commonwealth Health Laboratory, Darwin; the Western Australian Department of Public Health, Perth; and the Commonwealth Serum Laboratories, Melbourne.

singly or as mixed infections. All these investigations were done on patients with an obvious discharge and inflammation.

Large areas of the world, however, exist, mainly in East Asia and Australia, in which the trachoma is not obviously secondarily infected. Nothing is known of the bacteriology of these cases. In addition, the ophthalmic surveys of Western Australia carried out in 1953, 1954, and 1955 (Mann, 1957) suggested that staphylococcal infections (for example, styes and marginal blepharitis) were rare among tribal aboriginals and only began to occur where there was assimilation of the white and native races in a common way of life. In these areas (for example, the Kimberley District of Western Australia) the trachoma was also clinically much more severe and resembled that seen in Egypt.

It, therefore, seemed advisable to investigate the bacteriology of the conjunctival and nasal mucosa in areas where there was little or no contact with persons of European descent and where there were few or no clinical signs of infection. In the area chosen it is, however, remarkable that, although the eyes appear clean, there is often in children a severe mucopurulent nasal discharge. This does not appear to inconvenience the children and may be merely due to lack of nasal hygiene. The whole question of the distribution of pathogenic bacteria in these hot, dry, sparsely populated areas is an interesting one which has barely been touched. It is of importance in consolidating our knowledge of "trachome pur" since it will help us to decide whether the difference between this relatively innocuous disease and the serious "textbook trachoma" is merely one of presence or absence of secondary infection.

The investigation was undertaken by collaboration between the Public Health Department of Western Australia, the Northern Territory Medical Service, and the Commonwealth Serum Laboratories. The region selected was Arnhem Land (an aboriginal reserve not open for settlement) including Groote Eylandt, an isolated large island in the Gulf of Carpentaria to the east of Arnhem Land.

A preliminary survey of the trachoma incidence in the area in 1956 (Flynn, 1957) revealed an incidence of 74 percent in Groote Eylandt and 83 percent in Roper River on the adjacent mainland. Of 530 persons examined in these two places, 414 of them had trachoma but only two persons were blind (in one eye only) from the disease; 14 showed corneal involvement sufficient to diminish visual acuity but the rest had only very mild "trachome pur." No epidemics of mucopurulent conjunctivitis had been reported. This, therefore, seemed a suitable area for investigation of the bacteriology of the aboriginal eye.

In June, 1957, therefore, we proceeded by a Commonwealth Health Department plane from Darwin to Groote Eylandt carrying with us apparatus for the taking of cultures, conjunctival scrapings, and so forth. We cultured 41 children there of whom three had no clinical trachoma and two were prefollicular and doubtful, showing only papillary hypertrophy. Later we visited Goulburn Island off the north coast of Arnhem Land where we cultured five children who had had a course of sulfa treatment a few weeks before (all of them had subsiding trachome pur), and Oenpelli on the coast of Arnhem Land where we took cultures from six persons all of whom had mild trachome pur. None of these 52 aborigines had regular contact with white persons. In addition we investigated 14 children in the Native Settlement in Darwin where there is close contact with the European population. One of these children had impetigo. All had mild trachoma.

Clinical features

The salient clinical features of trachoma as observed in 52 Northern Territory aborigines on whom bacteriologic investigations were performed are shown in Table 1. The disease was in the early active stage in 42 patients. Apart from slight ptosis of the

upper eyelids in some cases, there was no external sign of infection and patients were free of symptoms. The tarsal conjunctiva was thickened, reddened, and granular. Typically the tarsal conjunctiva showed engorgement of the subepithelial capillaries, with consequent papillary hypertrophy, and, in addition, aggregations of inflammatory cells in follicular formation. These follicles showed central necrosis. Later the necrotic tissue became replaced by fibroblasts giving rise to fine scar tissue. Epithelial infiltrates of the upper part of the cornea were present with fine vessels extending usually not more than three mm. from the limbus. In only two cases was this pannus significant enough to produce slight blurring of vision, that is, it extended to the pupil area. We used the clinical classification endorsed by the Expert Committee on Trachoma of the World Health Organization namely:

Stage A. Active and infectious. Papillary hypertrophy follicles and epithelial corneal infiltrates.

Stage B. Healed by scarring without impairment of sight. No follicles.

Stage A-B. Both follicles and scars present.

Stage C. Healed with scarring of the cornea sufficient to impair sight.

Stage D. Certifiably blind from trachoma (for example, corneal scars, trichiasis, shrinkage of the conjunctiva, and so forth).

This is more satisfactory than the MacCallan classification as the latter makes no distinction between Stages B, C, and D which are all in MacCallan Stage 4 though not at all similar clinically.

We found 42 patients in Stage A, 20 in A-B with both follicles and scars, three healed completely with fine lid scars only (stage B), and one corneal scarring sufficient to impair sight (stage C). No patient was blind from trachoma or its complications.

We considered that Stage A was the most likely to be infectious and these cases were chosen for attempted culture.

The clinical features of trachoma in the Northern Territory correspond closely to those found in heavily infected areas of the Middle East where these latter cases are uncomplicated by infection with pathogenic bacteria (Thygeson, 1952). However, the course of the disease is more benign in Australia than in the Middle East and in the present series complete healing with minimal scar tissue formation, no entropion, and virtually no corneal involvement was the rule.

Bacteriologic investigations

Swabs were taken from the lower fornix, from the lid margin, and in most cases from the nostrils also. Five or six swabs were taken from each case. The swabs were smeared immediately on blood-agar plates. The plates were held at passenger-cabin temperature during transfer to the laboratory in Darwin where they were incubated for 48 hours at 37°C. In the case of Groote Eylandt, the plates were not incubated for 24 hours after inoculation, but the plates obtained at Oenpelli and Goulburn Island were incubated eight hours or less after inoculation. The necessity of working on open verandas greatly increased the risk of contaminating plates during inoculation in the field.

The results of bacteriologic examination of the conjunctival and nasal mucosa of 66 aborigines, 63 of whom had clinical trachoma, are shown in Table 1. The low incidence of common pathogens is most striking. The virtual absence of Staph. aureus from the noses of these aboriginal children is in sharp contrast to the relatively frequent occurrence of this organism in a comparable group of white children. Styes were encountered rarely in these aboriginal children, and this correlates well with the low incidence of infection with pathogenic staphylococci (table 2).

The presence of B. hemolytic streptococci is of interest as acute tonsillitis seems uncommon but rheumatic fever relatively com-

TABLE 1

CLINICAL FINDINGS IN 66 TRACHOMA PATIENTS

Clinical Signs	Follicles	Infiltrates	Papillae	Vessels	Scars	Herbert's Pits	Pannus
	61	28	24	15	24	10	2

Stage of Disease	A	A–B	B	C	D
	42	20	3	1	0

mon in these children. These organisms were found in the noses and not the eyes of the children and so appear unimportant in the eye pathology.

The coliform organisms were fairly common and were probably contaminants in the eye related to the unhygenic habits of so many of the children. It is possible that, knowing their eyes were to be examined, they put their fingers to them even more than usual just before the swabs were taken. The Proteus were probably of similar significance and possibly the Friedlanders, though the latter may be a normal inhabitant of the upper respiratory tract.

Tropical conditions and limitation of equipment that could be carried by air restricted the media which could be used for immediate plating of swabs to blood agar. Any colonies with characteristics suggestive of Koch-Weeks bacillus were transferred to heated blood media but none were shown to be Koch-Weeks organisms. The use of blood agar exclusively for the primary isolation of organisms in the field must be remembered when interpreting the results.

These results show only the bacterial flora present in June, 1957. However, the absence of outbreaks of conjunctivitis is evidence that bacterial pathogens are rare.

It is of some interest that the children at Bagot who are all in close contact with the white population of Darwin were free from pathogens. These were a small group and the number was further reduced by contamination of some of the plates.

The results show, in summary, a low incidence of conjunctival pathogens and a surprisingly low incidence of nasal carriers of Staph. aureus in these native children.

VIRUS STUDIES

Scrapings of upper lid conjunctiva were obtained for virus studies from 38 patients with trachoma, of whom 26 lived at Groote Eylandt, six at Oenpelli, and six at Goulburn Island (table 3). These scrapings which contained epithelial cells moistened by tears were dispersed in mixture 199 containing five-percent inactivated (56°C. for 30 minutes) calf serum. Aliquots of cell suspension were inoculated into stationary culture tubes of Hela cells and human amnion cells maintained in mixture 199 plus five-percent calf serum. In addition, plasma clot cultures on coverslips were made from aggregates of epithelial cells of all patients examined. These cultures were maintained in mixture

TABLE 2

RANGE OF BACTERIA ENCOUNTERED IN 66 PATIENTS EXAMINED

Site Examined	S. albus	Coliform	Nonhemolytic Streptococci	Gram-negative cocci	Friedlander's Bacillus	S. aureus	B. hemolytic Streptococci
Fornix	28	9	10	0	2	2	0
Lid	39	8	9	0	1	3	0
Nose	24	9	7	30	1	0	3

TABLE 3

VIRUS STUDIES ON TRACHOMA PATIENTS

District	Tissue Cultures Inoculated or Prepared			
	Hela	Amnion	Scrapings	Fornix
Groote Eylandt	21	26	26	8
Oenpelli	6	6	6	—
Goulburn Island	6	—	6	—

199 plus 20 percent inactivated human serum. The remainder of the suspension of conjunctival scrapings was sealed in ampoules and stored immediately on dry ice at −70°C. Small pieces of conjunctiva were excised from the upper and lower fornices of eight patients. These were attached to coverslips by chick plasma clot and maintained in mixture 199 plus 20-percent human serum.

Cultures of Hela cells (Syverton, et al., 1954) were prepared at the Commonwealth Serum Laboratories, Melbourne, six or seven days prior to inoculation and human amnion cultures (McLean and Cameron, 1957) were prepared 10 or 11 days before inoculation. They were transportd by air at passenger-cabin temperature to the Mission Stations, inoculated immediately after material was obtained from the patients, and transferred by air at cabin temperature to Melbourne. The cells were held at 37°C. in Darwin two or three days before and one or two days after inoculation in the field. In Melbourne, the cultures were incubated at 37°C. for 10 days in the case of Hela cells and 10 to 21 days in the case of amnion cells. When changes suggestive of infection with virus were observed in the cells, either the supernatant fluid was transferred immediately to other cultures, or the culture tube containing fluid was frozen and thawed, after which the fluid was inoculated into other cultures.

No transmissible agent was isolated from any of the conjunctival scrapings following inoculation of Hela or human amnion cultures. Although duplicate plasma clot cultures were prepared from scrapings from 38 persons, the cells from only one patient (No. 16) grew, and no change attributable to infection with virus was detected after 12 days' incubation. Of the eight preparations of fornix conjunctival explants, only two (No. 27 and No. 44) showed outgrowths of epithelial cells and no virus was isolated.

The upper lid conjunctiva of the right eyes of two rhesus monkeys were scarified with suspensions of scrapings from patients No. 20 and No. 27 respectively. No changes in the conjunctiva were observed during a 53-day observation period.

HISTOPATHOLOGY

Conjunctival scrapings from the upper eyelids of 51 of the patients on whom bacteriologic studies were performed were fixed on slides with ethyl alcohol and stained overnight with dilute Giemsa. Polymorphonuclear leukocytes were observed frequently, along with conjunctival epithelium, but mononuclear cells were less common. No inclusions resembling those described by Thygeson (1952) were seen in any of these smears.

SUMMARY

During June, 1957, bacteriologic and virologic investigations were undertaken on Australian aborigines affected with trachoma who lived at Groote Eylandt, Oenpelli, and Goulburn Island in the Northern Territory. The disease was almost entirely in the form of "trachome pur" with practically no cicatrization or pannus and a very low blindness rate.

Hemolytic staphylococci were found in the eyes of three patients out of 66 examined but they were not present in the noses. B hemolytic streptococci were found only in the noses of three patients, but not in their eyes. Friedlander's bacillus was found in the eyes and nose of one patient, and in another patient it was present only in the eye. This low incidence of bacterial pathogens is remarkable.

Attempts to isolate a virus from 38 patients using established cultures of human amnion and Hela cells and from explants of conjunctiva from patients were unsuccessful.

No viruslike inclusions were seen in stained smears of tarsal conjunctiva from 51 patients.

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ADDENDUM

After this article was prepared for publication we learned that trachoma virus has been isolated from Gambian trachomatous patients by L. H. Collier and J. Sowa (Lancet, 1:993, 1958), using inoculation of the six-day-old embryonated egg yolk sac. It is hoped that further attempts to isolate virus from Australian trachomatous patients by this technique will be undertaken shortly.

REFERENCES

Flynn, F.: Trachoma among natives of the Northern Territory of Australia. Med. J. Austral., **2**:269-277, 1957.

Mann, I.: Report on ophthalmic findings in Warburton Range natives of Central Australia. Med. J. Austral., **2**:610-612, 1957.

———: Ophthalmic Survey of the South West Portion of Western Australia. Government Printer, Perth, 1957.

———: Ophthalmic Survey of the Kimberley Division of Western Australia. Government Printer, Perth, 1954.

———: Ophthalmic Survey of the Eastern Goldfields Area of Western Australia. Government Printer, Perth, 1954.

McLean, D. M., and Cameron, D.: Aseptic meningitis due to infection with ECHO virus (type 9). J. Hyg., **55**:464-474, 1957.

Syverton, J. T., Scherer, W. F., and Elwood, P. M.: Studies on the propagation in vitro of poliomyelitis viruses. The application of strain Hela human epithelial cells for isolation and typing. J. Lab. & Clin. Med., **43**:286-302, 1954.

Thygeson, P.: In Viral and Rickettsial Infections of Man (T. M. Rivers, editor). Philadelphia, Lippincott, ed. 2, 1952, pp. 465-471.

SURGICAL TREATMENT OF ESOTROPIA*

ANALYSIS OF CASE MATERIAL AND RESULTS IN 315 CONSECUTIVE CASES

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The ideal goal in the treatment of esotropia is the prevention of amblyopia and the development of the best possible binocular vision or, at the very least, attainment of an acceptable cosmetic result.

During the 10 years from January, 1947, through December, 1956, 1,427 patients having esotropia were seen at the Cleveland Clinic; 315 of these patients received surgical treatment. The case material and the results are discussed in this report. No case in the 10-year interval has been omitted. An attempt has been made to classify all cases, and to present the preoperative and postoperative findings under each separate group of the classification.[1]

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METHOD OF STUDY

Three hundred and fifteen case histories of consecutive patients who underwent operation for esotropia were studied and sig-

nificant items from each were recorded. These items were: age of patient when first examined here; probable age of patient at onset of esotropia; past record of esotropia; family history; visual acuity and refractive error; preoperative deviation in prism diopters (including hypertropia when present); status of patient's fusion; presence or absence of anomalous retinal correspondence, head tilt, and nystagmus; status of oculorotary muscles (ductions, versions, and vergences); preoperative management; surgical procedure; postoperative deviation (usually measured from three to five weeks after surgery); postoperative treatment (for example, orthoptics, occlusion, glasses); deviation, status of fusion, refractive error, and visual acuity at last examination; and postoperative follow-up in years.

It must be remembered that the case histories represent the knowledge and memories of the patients, or their parents, in regard to some of the information, and that they are accurate or complete only insofar as any case history ever is exact.

Diagnosis of esotropia

In general, all patients were given the same diagnostic work-up. The history and visual acuity first were recorded. The history ideally included data concerning: the probable time of onset of the esotropia; the deviating, the fixating, or the dominant eye; the variability of the esotropia; the presence or absence of nystagmus, head tilt and diplopia; the effect of glasses (if worn) in reducing the esotropia; previous treatment if any; family history of heterotropia; the health of the mother during pregnancy, for example, whether or not she had rubella; the history of the mother's labor and delivery; the neonatal health of the patient; the development and growth of the patient; and the association of the onset of the strabismus with various precipitating factors, such as exanthematous diseases, fever, convulsions, trauma, prematurity, mental retardation.

The visual acuity was tested either with the standard Snellen chart at a distance of six m., or the illiterate "E" test, if possible. Children of four years usually were able to understand the "E" test, though it was appropriate for some who were younger or older. Some patients who originally were recorded as having an acuity of 6/9 as determined with the "E" test, later were found to have suppression amblyopia. This discrepancy is a recognized disadvantage of the "E" test.

The external examination was followed by a detailed study of the oculorotary muscles. Whenever possible, the deviation was measured by the cover test in the primary position at six m. and at 33 cm., with and without glasses. In cases of paralytic esotropia and those of associated hypertropia, the deviation also was determined in each of the cardinal positions. If the esotropia was obviated at six m. with glasses, but uninfluenced at near with the correction, then a +3.0D. sph. was incorporated into the correction at 33 cm. and the deviation again was measured. This helped in determining whether or not bifocals were indicated. The corneal reflex (Hirschberg's) test and/or the light-prism reflex test were given to children whose co-operation was insufficient for the cover test. The angle kappa was recorded only when it was positive. The magnitude of any vertical deviation was determined in the same manner as was the horizontal deviation. The relative near-point of convergence was measured with a ruler and muscle light.

The presence or absence of diplopia (physiologic or anomalous) was determined using red-green glasses. This test also was of value in determining the presence or absence of amblyopia, and in deciding whether or not the esotropia was monocular or alternating. The bifixation test was used in rapid alternators who had slight degrees of esotropia and in whom it was difficult to tell whether or not fusion was present. Duction studies were performed on each eye; these were followed by versions at near in the cardinal positions. The deviation was deter-

mined again on the major amblyoscope. The objective angle was measured first; then the subjective angle was determined. A difference between the two angles suggested the diagnosis of anomalous retinal correspondence. In such cases the angle of anomaly was determined and the anomalous retinal correspondence classified as harmonious or unharmonious. The degree of fusion was measured on the major amblyoscope.

Ophthalmoscopic examination was followed by other studies as necessary, for example, visual fields, neurologic examination. With few exceptions a cycloplegic refraction was performed on all patients.

Management of esotropia

The preoperative treatment varied according to age of the patient, visual acuity, refraction, magnitude of the esotropia, and other factors, for example, whether or not a functional result was anticipated. Glasses were prescribed for all patients in whom the visual acuity was improved or in whom the deviation was reduced. Bifocals often, but not always, were prescribed when a significant residual deviation remained at 33 cm. A paste-on type of bifocal was prescribed, fitted such that the upper margin of the segment split the pupil.

Treatment of suppression amblyopia, whenever possible, was by total 24-hour occlusion with an Elastoplast type of occluder. Atropine drops were used in infants who appeared to be habitually fixating with one eye, and in whom the differential could not be made between an alternating and a monocular esotropia.

The specific surgical procedure to be used depended on many factors, such as age of the patient, presence or absence of amblyopia, size and variability of the esotropia, whether or not the esotropia was alternating or monocular, influence on the esotropia by glasses and general anesthesia, presence or absence of significant hypertropia, and whether the esotropia was greater at six m. than at 33 cm. or greater at 33 cm. than at six m. One or more of the following surgical procedures were used:

1. Bimedial rectus recession.
2. Monocular medial rectus recession.
3. Recession of the medial rectus and resection of the lateral rectus.
4. Recession of the medial rectus and tuck of the lateral rectus.
5. Resection or tuck of a lateral rectus.
6. Tenotomy of the medial rectus.

A combined horizontal and vertical procedure was done in only three cases. In monocular esotropia, the usual procedure on the amblyopic eye was a recession of the medial rectus along with a resection or tuck of the lateral rectus. In alternating esotropia, the usual procedure was a bimedial rectus recession, but, depending on the size of the strabismus, a single medial rectus was recessed (smaller degree) or a recession-resection of the nondominant eye (larger degree) was done. When an accommodative component was present, either in alternating or in monocular cases, less surgery was done. Cases with convergence excess were more prone to have had a bimedial recession, whereas those with divergence insufficiency were more likely to have had a single medial rectus recessed and a tuck or resection of the lateral rectus. All operations were performed under general anesthesia.

One should not expect the immediately postoperative deviation to remain the same in all instances. Often the deviation that was present six weeks postoperatively was different from the deviation that was present two years later. In some patients the sixth-week postoperative deviation was equal to the preoperative deviation, and subsequent cosmetic results were excellent; in others the deviation remained in status quo. In other patients, excellent sixth-week postoperative results culminated in exotropia. It was rare for an immediately postoperative severe exotropia spontaneously to eventuate in fusion or cosmetically straight eyes. If the patient had reasonably good vision without his glasses and if the correction influenced the

strabismus little, if at all, then they were discarded. Before formal orthoptics were begun in any patient it was necessary that he have equal or near-equal vision in each eye (at least 6/15) and reasonably well-aligned eyes. An intelligent, co-operative patient, along with understanding parents, was a distinct advantage; further, a functional result must have been anticipated. Most of the patients who received this treatment were more than six years old. The length and specific type of training varied among individuals; one patient was treated for two years. The type of exercise depended on whether or not the patient's problem was anomalous retinal correspondence, suppression, or poor fusional amplitudes.

Case material

Sex and age of patients when first seen (table 1)

Of the 315 patients, 151 (47.9 percent) were males and 164 (52.1 percent) were females. The ages of the patients ranged from 4.5 months to 59 years. One hundred and fifty-two (48.2 percent) of the patients were six or more years of age and were not the most desirable candidates for a functional cure or successful treatment of amblyopia if present. The interval usually was long from the stated onset of the esotropia to the initial examination here.

TABLE 1

Approximate age of patient when first seen

Age (yr.)	No. of Patients
<1	8 (youngest 4½ mo.)
1	16
2	32
3	43
4	30
5	34
6	22
7	14
8	12
9	10
10	9
11–20	53
21–30	23
31–40	4
41–59	5
Total	315

TABLE 2

Approximate age (from history) at onset of esotropia

Age (yr.)	No. of Patients
Birth	75
<1	45
1	38
2	49
3	37
4	21
5	12
6	4
7	3
8	4
>8	6 (oldest 23)
Unknown	21
Total	315

Age at onset of strabismus (table 2)

In 265 (84.1 percent) of the patients the strabismus was said to have begun during the period from birth to the age of four years, inclusive, and in 158 (50.1 percent) during the period from birth to the age of one year inclusive. In 119 (37.7 percent) instances, the esotropia was said to have begun in the second, third, fourth, or fifth year.

Other data

Significant data that were tabulated were previous treatment of any kind, the family history, and possible etiologic, associated, or precipitating factors. In nearly all cases, the patient was or had been wearing glasses. Thirteen patients (4.1 percent) had undergone one or more surgical procedures, nine (2.8 percent) had had "orthoptics," and 35 (11.2 percent) had had some form of occlusion therapy. One hundred and two patients (32.3 percent) had a positive family history (table 3); the type of strabismus of the relatives rarely was known. One hundred and fifty-seven patients (49.9 percent) had a negative family history for strabismus. There was no mention of the family history in 56 cases (17.8 percent).

TABLE 3

FAMILY HISTORY OF ESOTROPIA

Family Members Affected	No. of Patients
Father, mother, or sibling	62
Grandparent	8
Aunt or uncle	17
First cousin	15
None	157
Not listed on chart	56
Total	315

The cause of the esotropia, cited by the patient or his parents, was recorded in 107 (33.9 percent) of the case histories. Associated with the onset of the esotropia in 17 cases (5.4 percent) was indirect trauma, for example, automobile accident, fall from a window. Four patients (1.3 percent) had direct trauma to the eye with a resultant corneal scar, complicated cataract, or optic atrophy; three (0.9 percent) had a congenital cataract of one or both eyes. Ten (3.2 percent) were born prematurely; six (1.9 percent) were born by cesarean section; and the birth of 13 (4.1 percent) involved a difficult labor. In 28 cases (8.9 percent), the esotropia occurred during or after one of the exanthematous diseases, pertussis, a cold, or poliomyelitis (one case). Other associated conditions included mental retardation, epilepsy, cerebral palsy, congenital anomalies of other parts of the body, and a cerebellar tumor. Some cases were thought to have been precipitated by tonsillectomies. One case of esotropia was thought to have been caused by fright.

CLASSIFICATION

The classification used was that reported by Costenbader.[1] Table 4 lists the number of cases that ocurred in each group and subgroup of the classification. Accurate classification of many cases was next to impossible, and differentiation between a monocular and an alternating esotropia often was difficult. In some instances characteristics of more than one subtype would appear. A few had the deviational features of one of the subclassifications, but the refractive error did not approach that expected. Despite these difficulties, we believe that classification of cases is of great importance in the evaluation of the results of treatment as a basis for selecting treatment and determining prognosis in future cases.

Eighty-six cases (27.3 percent) were thought to be of the mixed type, that is, had an accommodative and a nonaccommodative component. Glasses would reduce a significant part but not all of the esotropia and in some cases at distance but none at near.

There were only 12 cases (3.8 percent) of

TABLE 4

CLASSIFICATION (AFTER COSTENBADER)

Type of Esotropia	No. of Patients			Percentage
	Alternating	Monocular	Total	
A. Comitant esotropia				
1. Accommodative				
a. Refractional	0	0	0	0
b. Hypoaccommodative	9	7	16	5.1
c. Hyperkinetic	0	0	0	0
2. Nonaccomodative				
a. Tonic				
(1) Later onset	60	39	99	31.5
(2) Congenital	46	39	85	26.9
(3) Congenital myopia	7	3	10	3.2
b. Mechanical	0	0	0	0
3. Mixed	62	24	86	27.3
B. Noncomitant esotropia				
1. Anatomical	2	0	2	0.6
2. Paretic	5	5	10	3.2
C. Unable to classify (optic atrophy, cataract, corneal opacity)	2	5	7	2.2
Total	193	122	315	100

TABLE 5

AMBLYOPIA IN MONOCULAR ESOTROPIA

Degree of Amblyopia	Age, Years	
	Six Years or Less	More than Six Years
No central fixation	7	12
6/60 or less, but able to fixate	24	19
6/30	7	10
6/21	2	9
6/15	7	2
6/12	4	0
6/9 (later found to have more severe amblyopia)	3	0
No visual acuity because of age—later amblyopia	16	0
	70	52
Total	122	

noncomitant esotropia. Of the paretic type, each had a palsied sixth nerve. The two anatomic cases consisted of one patient with Duane's syndrome and one with strabismus fixus. The seven cases (2.2 percent) of cataract, corneal scar, or optic atrophy were listed separately.

There were 193 alternating esotropias (61.2 percent) in the series; of these, 78 had no preference for either eye, 107 preferred to use one or the other eye, and eight patients alternated only at distance or at near. Forty-seven patients were cross-alternators, but this tendency did not appear to influence the surgical result.

One hundred and twenty-two patients (38.8 percent) were thought to have monocular esotropia. Most of these patients had suppression amblyopia (table 5), the most severe complication of strabismus. This is the disabling end-result of suppressed macular vision during the plastic, formative years from birth to the age of about six years. Thus it is that early treatment is the essence of management of these cases. These patients were divided into two groups, those aged six years or less, and those older than six years. The age of six years was selected because of the general feeling that the development of visual acuity and binocular habits usually terminates about then, also because the results of treatments of amblyopia can be better evaluated. One hundred and eighty-five patients (58.7 percent) were six years old or less when first examined. Of these, 70 (22.3 percent) had some degree of amblyopia and 19 of them had improvement with treatment. Three patients thought to have 6/9 vision (illiterate "E") in each eye when first examined were later found to have amblyopia. Sixteen patients, part of a group in which testing of visual acuity was not possible at their first visit, later were found to have amblyopia. Table 6 shows the results of the treatment of suppression amblyopia in this group. The treatment was administered either preoperatively or postoperatively, or at both periods, and for the most part consisted of total 24-hour Elastoplast occlusion. Occlusion therapy was unsuccessful in 34 percent of the patients less than six years of

TABLE 6

TREATMENT OF AMBLYOPIA

Therapy	Number of patients			
	Six Years of Age or Less		More than Six Years of Age	
Occlusion				
Treated preoperatively and/or postoperatively—substantial improvement	19		5	
Treated—little or no improvement	24		5	
Not treated with occlusion	27		42	
Improved spontaneously with glasses		7		1
Had had previous treatment—failure		20		41
Total	70		52	
Grand total	122			

age who had amblyopia. Twenty-seven patients (8.5 percent) aged six years or less were not treated with occlusion. Most of these had had previous unsuccessful attempts at occlusion. Some patients had spontaneous improvement of their amplyopia with constant wearing of glasses.

Preoperative deviation

Table 7 lists the preoperative deviation in prism diopters as determined with the cover test when that test could be done. When it was impossible to perform this test, the light-prism reflex and Hirschberg's test were used. Unless otherwise indicated in the table, the deviation recorded was that measured without glasses at six m. One hundred and forty-eight patients (46.8 percent) had a hypertropia of five prism diopters or greater at the same distance that the horizontal deviation was measured. Thirty-six of these had bilateral hypertropia. This is significant because, as will be seen later, an operation on the horizontal recti only, in most instances, does not correct fully the vertical error.

Refractive error

The hyperopic component of the refraction is tabulated in Table 8. Many patients had a small cylindrical error, but this was not entered in the table. Only when the cylinder was larger than 2.0D. (plus or minus) was the case listed as one of astigmatism. Values from −1.0 to −10.0D. of myopia were found in the patients with congenital myopic nonaccommodative esotropia.

There was no direct correlation between the magnitude of the hyperopia and the deviation. In some instances a +1.0D. sph. would reduce the deviation by from 15 to 25 prism diopters; in others, hyperopia was as high as +9.0D., but the esotropia was

TABLE 7

Preoperative deviation

No. of Cases	Classification	Deviation in Prism Diopters				Presence of Hypertropia >5 prism diopters	
		10–19	20–39	40–50	60 or >	R. or L.	Bilateral
	Alternating comitant esotropia						
9	Hypoaccommodative	2*	6*	1*	—	1*	2*
60	Nonaccommodative, tonic, late onset	3	23	28	6	17	2
46	Nonaccommodative, tonic, congenital	—	19	22	5	19	11
7	Nonaccommodative, congenital, myopia	—	2	4	1	2	1
62	Mixed	15†	29†	14†	4†	18†	5†
	Noncomitant						
2	Anatomic	—	1	1	—	1	1
5	Paretic	1	3	1	—	—	—
2	Unable to classify (congenital cataract)	1	—	1	—	1	—
	Monocular comitant esotropia						
7	Hypoaccommodative	2*	4*	1*	—	2*	—
39	Nonaccommodative, tonic, late onset	2	16	17	4	14	—
39	Nonaccommodative, tonic, congential	4	20	14	1	19	7
3	Nonaccommodative, congenital, myopia	—	1	1	1	3	—
24	Mixed	9†	7†	6†	2†	11†	7†
	Noncomitant						
5	Paretic	—	2	3	—	2	—
5	Unable to classify (cataract, optic atrophy, corneal scar)	2	1	1	1	2	—
		41	134	115	25	112	36
		315				148	

* Deviation measured with glasses at 33 cm.
† Deviation measured with glasses at 6 m.

TABLE 8

REFRACTIVE ERROR

Classification	0.25 to 1.0D.	1.25 to 2.0D.	2.25 to 3.0D.	>3.0D	Myopia	Astig-matism	Antime-tropia	Not on chart
Alternating								
Hypoaccommodative	3	1	2	2	1	—	—	—
Nonaccommodative, tonic, late onset	14	21	8	5	1	5	3	3
Nonaccommodative, tonic, congenital	13	6	4	2	—	9	5	7
Nonaccommodative, congenital myopia	—	—	—	—	7 (−1.0 to −10.0D.)	—	—	—
Mixed	7	17	7	25	—	2	4	—
Anatomic	—	—	1	—	—	—	—	1
Paretic	—	—	1	—	—	2	1	1
Unable to classify (congenital cataract)		—	—	2	—	—	—	—
Monocular								
Hypoaccommodative	1	2	—	1	—	—	3	—
Nonaccommodative, tonic, late onset	10	5	6	6	2	4	6	—
Nonaccommodative, tonic, congenital	12	8	6	1	1	1	6	4
Nonaccommodative, congenital, myopia	—	—	—	—	3	—	—	—
Mixed	—	5	8	10	—	—	1	—
Paretic	1	—	—	—	1	1	1	1
Unable to classify (cataract, optic atrophy, corneal scar)	—	—	1	—	1	1	1	1
Total	61	65	44	54	17	25	31	18
Grand Total				315				

not reduced a single diopter with the correction. Significant astigmatism predominantly occurred in nonaccommodative tonic esotropia. Antimetropia (anisometropia) occurred in 31 cases (9.8 percent) and when an amblyopic eye was present in these it had the greater refractive error of the two eyes in all instances.

ANOMALOUS RETINAL CORRESPONDENCE AND NYSTAGMUS

Anomalous retinal correspondence was present in 44 patients with alternating esotropia and in 33 with monocular esotropia. Of these 77 patients (24.5 percent), only five had a harmonious anomalous retinal correspondence, that is, the angle of anomaly was equal to the objective angle of the esotropia.

Oculorotary nystagmus was present in 10 cases (3.2 percent) and five of these were in the alternating congenital nonaccommodative group.

SURGICAL PROCEDURE

Table 9 lists the surgery performed and the number of cases requiring two or more operations. Thirty-two patients (10.2 percent) had more than one surgical procedure. A total of 347 operations was performed and of these, 122 (34.1 percent) operations were on patients eight years of age or more.

RESULTS

To tabulate properly and fairly the end-results of any kind of treatment is sometimes an uneasy task. This is especially true of surgical treatment of esotropia. Normal vision in the presence of third-degree fusion and normal amplitudes of vergence is the best result. Then follow second- and first-degree fusion, small degrees of esotropia,

TABLE 9

SURGICAL PROCEDURE AND POSTOPERATIVE ORTHOPTICS

Classification	Bimedial Recession	Recess-Resect	Recess-Tuck	Recess Medial Rectus	Resect or Tuck Lateral Rectus	Tenotomy Medial Rectus	Required Two or More Operations	Orthoptics, Postoperative
Alternating								
Hypoaccommodative	4	2	—	3	—	—	1*	1
Nonaccommodative, tonic, late onset	33	16	4	9	2	—	4	16
Nonaccommodative, tonic, congenital	20	11	5	10	7	—	7	7
Nonaccommodative, congenital, myopia	4	2	—	—	1	—	—	1
Mixed	27	21	5	11	3	2	7	15
Anatomic	1	1	—	—	—	—	1*	—
Paretic	2	1	1	1	—	—	—	—
Unable to classify (congenital cataract)	—	1	—	1	—	—	—	—
Monocular								
Hypoaccommodative	—	2	—	5	—	—	—	2
Nonaccommodative, tonic, late onset	2	31	2	4	2	1	4†	8
Nonaccommodative, tonic, congenital	2	26	5	7	3	1	5	10
Nonaccommodative, congenital, myopia	—	2	—	2	—	—	1	—
Mixed	3	14	1	5	1	—	—	5
Paretic	—	4	—	2	—	1	2	—
Unable to classify (cataract, optic atrophy, corneal scar)	—	2	1	2	—	—	—	—
Total	98	136	24	62	19	5	32	65

* Additional operation for hypertropia.
† 1 patient operated on for postoperative exotropia.

moderate esotropia, severe esotropia, exotropia, persistent postoperative diplopia, and "horrors fusionis." No cases of the last two have occurred.

Twenty-three patients (7.3 percent) were not seen again after their discharge from the hospital; most of them returned to the care of the referring physician.

The question of functional results is an important one. It would be ideal to say that most patients will develop fusion after surgery, but realization of this goal often is not attained. A functional result (table 10) is one in which the patient secured binocular use of his eyes; this occurred in 32 patients (10.2 percent) postoperatively. Of these, 17 were able to recognize stereopsis. Statistically this figure can be raised depending upon the criteria for selection of cases. For example, if we exclude all patients more than 10 years of age, and those less than 10 with an amblyopic eye at the time of operation, the number of cases drops to 130, the functional cures number 26, and the resultant percentage is 20.

There was a variable interval between time of surgery and the development of fusion. This depended on many factors, such as demonstration of fusion at the objective angle preoperatively, age at the time of surgery, and presence or absence of hypertropia or anomalous retinal correspondence. Those who developed fusion most rapidly when the eyes were straightened surgically were the patients in whom one preoperatively could elicit fusion with relative ease on the major amblyoscope. It is significant that no patients thought to have congenital, nonaccommodative, myopic esotropia developed fusion. Only one patient of the 85 of those thought

to have congenital nonaccommodative, tonic esotropia developed fusion and this was only first degree. These facts are significant and demonstrate the difficulty a patient has in developing something that he has never experienced.

Postoperative orthoptics appeared to contribute directly to the development of fusion in one-third of those patients who had a functional result. Most patients, excluding those with no central fixation, were tested on the major amblyoscope postoperatively to determine their fusion. If a patient had this, then he might be a good candidate for formal orthoptics. It must be remembered that the fusional status of a patient's eyes on the major amblyoscope may be different from that of his everyday casual seeing. Glasses often were necessary to maintain a patient's fusion. A few patients were able to fuse only at six m. or at 33 cm. Some had exceedingly low amplitudes of vergence. Fusion was listed by degree (first, second, third).

Good cosmetic results are important in the surgery of esotropia. Often it is the prime factor in the decision to operate upon a patient, and many patients voluntarily see the surgeon just for that purpose—fully realizing that the visual acuity of an amblyopic eye will not be changed. The psychologic aspects of this particular problem often overshadow the status of the visual acuity and the binocular habits of the patient. Patients with disfiguring strabismus deserve the benefits of surgery, and it is important that every means available is afforded them to secure reasonably straight eyes. However, these cases are not to be taken lightly because of the complications that may occur. Table 10 lists the results by the individual groups in the classification. Cosmetic results were not graded on a scale from excellent to bad; rather, the actual measurement was listed. Even though some patients had a large residuum of esotropia, they were happy with the result because their appearance postoperatively was cosmetically acceptable. Still, it is necessary to tabulate results as objectively as possible, and the preferred manner is to record the actual deviation in prism diopters or degrees.

Eighty-seven patients (27.6 percent) at their last visits had deviations that varied from an exotropia of five prism diopters to an esotropia of nine prism diopters. Seventy-three patients (23.2 percent) at their last visits had deviations that varied from 10 to 19 prism diopters of esotropia. Forty-four patients (14.0 percent) had final deviations varying from 20 to 29 prism diopters of esotropia. Twenty-eight patients (8.9 percent) had final deviations of 30 or more prism diopters of esotropia. Of these 28 patients, 16 were thought to have congenital, nonaccommodative, tonic esotropia. This is a significant value. The postoperative and preoperative deviations were measured at the same distances. For a small number of patients the test used to measure the deviation postoperatively was not the same as that which was used preoperatively. Horizontal surgery alone reduced associated hypertropia to an insignificant value (less than five prism diopters) in eight of 142 patients.

Exotropia is an undesirable complication of surgery for esotropia. Anyone having an exotropia of six prism diopters or greater was listed as having this complication; 28 patients (8.9 percent) had this complication. Of these, 22 had exotropia in the range of six to 20 prism diopters; six had more than 20 diopters, the highest being 40 prism diopters. The condition was seen in patients having poor fixation in an amblyopic eye, or variability in the magnitude of the deviation; also in patients whose esotropia had an early onset with little, if any, accommodative component. Of significance is the fact that the condition did not develop in any of the patients with congenital myopia. It is important to watch the relative near-point of convergence postoperatively because a recessed near-point of convergence (greater than 80 mm.) may be the first sign of warning that exotropia will eventuate (table 10).

TABLE 10

SUMMARY OF RESULTS AND OTHER DATA

Classification	Number of Patients: Results: Functional (fusion by degree) 1	2	3	Cosmetic, in Prism Diopters (measured at six m. without glasses unless otherwise indicated) X^T5 to E^T9	E^T10 to E^T19	E^T20 to E^T29	$E^T>30$	X^T6 to X^T20	$X^T>20$	No Follow-Up	Recess NPC 80 mm. or Greater	Reversion of Esotropia to Preoperative Deviation or Greater	Average Follow-up (yr.)
Alternating													
Hypoaccommodative	—	1	2	1*	—	2*	—	2*	1*	—	2	1	4.5
Nonaccommodative, tonic, late onset	—	2	5	16	16	11	3	4	—	3	18	5	2.7
Nonaccommodative, tonic, congenital	—	—	—	13	9	8	10	2	1	3	14	5	2.5
Nonaccommodative, congenital myopia	—	—	—	2	2	1	1	—	—	1	3	1	2.7
Mixed	1PS	8	5	16†	17†	8†	3†	1†	1†	2	14	8	3.8
Anatomic	—	—	1	1	—	—	—	—	—	—	—	—	1.8
Paretic	—	—	—	1	1	1	—	—	1	1	3	—	3.6
Unable to classify (congenital cataract)	—	—	—	—	1	1	—	—	—	—	1	1	—
Monocular													
Hypoaccommodative	—	—	—	1*	3*	1*	1*	—	—	1	2	4	2.5
Nonaccommodative, tonic, late onset	—	1	1	13	10	5	2	3	—	4	11	4	2.1
Nonaccommodative, tonic, congenital	1	—	—	8	8	4	6	4	2	6	9	10	2.1
Nonaccommodative, congenital myopia	—	—	—	1	1	1	—	—	—	—	2	—	3.6
Mixed	—	1	2	11†	4†	—	1†	3†	—	2	9	3	2.8
Paretic	—	—	1	1	—	—	1	2	—	—	—	—	2.6
Unable to classify (cataract, optic atrophy, corneal scar)	—	—	—	2	1	1	—	1	—	—	3	—	2.3
Total	2	13	17	87	73	44	28	22	6	23	91	42	(Av.2 64)

PS-Periodic esotropia.
* Deviation measured with glasses at 33 cm.
† Deviation measured with glasses at 6 m.

Ninety-one patients (28.8 percent) postoperatively had their relative near-point of convergence recessed to 80 mm. or more.

It is discouraging to see an excellent cosmetic result revert to the preoperative deviation. This occurred in 42 (13.4 percent) patients (table 10). Eight of these were known to have had anomalous retinal correspondence preoperatively and/or postoperatively. Among the factors contributing to this complication are: (1) too-conservative surgery, (2) anomalous retinal correspondence, and (3) postoperative adhesions at the site of surgery. Again the congenital, nonaccommodative, tonic cases developed this complication more frequently than others. Most frequently this was a slow process, but in some patients it reverted within two months. Despite three operations, one patient continued to revert to the original esotropia.

The average follow-up in years, as listed, is self-explanatory (table 10). The length of the follow-up is important because the longer it is, the more significant the statistics become. Eventually, the deviation in most cases becomes stabilized; however, the esotropia of some remains variable for long periods of time. This particular aspect of the problem was not studied in detail.

Summary

An analysis and results of surgery of 315 consecutive cases of esotropia are presented. In 10 percent of the patients some degree of fusion developed postoperatively. Cosmetic results were tabulated in prism diopters: 51 percent of the patients had a final deviation that ranged from an exotropia of five prism diopters to an esotropia of 19 prism diopters, 14 percent had a residual esotropia that varied from 20 to 29 prism diopters, and nine percent had greater than 30 prism diopters. Exotropia developed in nine percent of patients. There was no follow-up in seven percent of the cases. In 13 percent there was reversion to the original preoperative deviation. The average follow-up was 2.6 years. Postoperative formal orthoptics was thought to contribute to the functional result in 33 percent of patients in whom fusion developed. With surgery, one can only attempt to align the eyes; it does not of itself purport to keep them where they were placed. Where they eventually stabilize depends on the particular idiosyncrasies of each individual patient, and one cannot always forecast the long-term outcome.

2020 East 93rd Street (6).

Reference

1. Costenbader, F. D.: The management of convergent strabismus. In Strabismus Ophthalmic Symposium (James H. Allen, editor). St. Louis, Mosby, 1950, p. 476.

THE PATHOLOGY OF THE TRABECULAR MESHWORK IN PRIMARY OPEN-ANGLE GLAUCOMA*

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Primary open-angle glaucoma is associated with an increased resistance to aqueous outflow. This increase in resistance is organic in nature, for it has been shown by perfusion studies[4, 14, 17] that it is essentially the same after the eye is enucleated as before. There is no doubt that this higher resistance to aqueous outflow lies in the area of the filtration angle, but its exact site is still disputed.

Only a small number of eyes with early open-angle glaucoma have been described histologically because of the difficulty of ob-

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taining specimens before synechias and other late changes have occurred. However, a number of workers have described pathologic changes in the trabecular meshwork of such eyes.

In pathologic studies of four enucleated glaucomatous eyes, Teng, Paton, and Katzin[28] found degenerative changes in the entire area of the drainage angle, but they were most marked in the external part of the trabecular meshwork. These changes consisted of degeneration of collagen tissue and proliferation of the endothelium of Schlemm's canal. In their studies, they found similar degenerative changes in 10 percent of eyes from the eye-bank of people over 50 years of age; but these pathologic alterations were never seen in eyes of people under the age of 20 years. They suggested that open-angle glaucoma is due to a primary degeneration in the connective tissue of the filtration angle.

In another study of eyes with primary open-angle glaucoma, Theobald and Kirk[29] found thickening and sclerosis of the trabeculae and collector channels in the sclera. In some of their illustrations they noted narrowing of Schlemm's canal and a spongy edema of the external trabeculae, with deposition of pigment in this area. Their studies led them to believe that hypertrophy and sclerosis of collagenous fibers of the sclera associated with aging compresses and narrows intrascleral veins, producing glaucoma.

Maggiore,[24] François and his colleagues,[13] and others have found thickening and sclerosis of the trabecular fibers in cases of primary glaucoma.

Ashton, Brini, and Smith[1] found artefactual adhesions in Schlemm's canal in 25 percent of normal eyes. They warn against misinterpreting as pathologic, variations in the microscopic anatomy of the meshwork which may be found in normal eyes.

That the trabecular meshwork is the area producing the obstruction has been advocated by some writers.[16,23] It has recently been pointed out that the anatomy of the meshwork is incompletely visualized in ordinary pathologic studies because the meridional sections which are made allow only an unsatisfactory view of the meshwork.[19] Transverse and, particularly, tangential sections of the meshwork are necessary to appreciate its anatomy fully.

Believing that such sections are equally essential for a study of the pathologic changes in the trabeculae, a study has been made in transverse, meridional, and serial tangential sections of eyes with primary open-angle glaucoma, and of eyes with a variety of types of secondary glaucoma with open filtration angles.

Striking degenerative changes were found in the trabecular meshwork of eyes with primary and with secondary glaucoma. The character of these changes suggests a new conception of the pathogenesis of primary open-angle glaucoma.

Materials and Methods

The trabecular meshwork of 16 eyes with primary open-angle glaucoma and of 11 eyes with a variety of types of secondary glaucoma with open filtration angles were studied.

Most of the material was obtained from the Armed Forces Institute of Pathology, which co-operated in the study by forwarding to me unused wet tissue from eyes with open-angle glaucoma.

The material forwarded by the Armed Forces Institute of Pathology consisted of one or more meridional sections of each eye and the remaining wet tissue of the eye not used in making the original block. From this tissue enough material was usually obtained to prepare tangential sections of the meshwork in situ from three different quadrants of the eye.

In every case meridional sections as well as tangential sections were studied.

The tangential sections were prepared in a manner described in a previous paper.[9] Because of the unavoidable slight curvature

occurring in the tissue studied, oblique and transverse sections of the trabecular meshwork could be seen in some of the sections in almost every eye. Alternating slides of the flat sections were stained with Masson's trichrome stain. The remainder of the slides were stained with a variety of stains, including Verhoeff's elastic tissue stain, hematoxylin and eosin, Mollier's elastic, and periodic acid-Schiff. The Masson stain was found to be particularly helpful. Sclerosis and an increase in collagen are strikingly demonstrated with this stain in a deep blue color. The nuclei and other components of the meshwork also show up well with the Masson's stain. From 10 to 150 sections of the trabecular meshwork were examined in each glaucomatous eye, a total of over 2,000 sections of the trabecular meshwork of glaucomatous eyes. These sections were compared with similar sections made from normal eyes reported previously.[9]

All of the cases of both primary and secondary glaucoma were characterized by open angles. The histories, as given, were carefully studied (table 1). In 12 of the 16 eyes with primary open-angle glaucoma, the opposite eye was known to be involved by the same disease. In the other four cases, either the condition of the other eye was unknown or it was said to be uninvolved. In these cases, however, when the rest of the history of the disease was considered along with the pathologic sections showing the open angles, deep chamber, and cupped discs, there was little room for doubt of the diagnosis of primary open-angle glaucoma.

Some of the eyes were completely blind before they were removed; others were removed much earlier in the course of the disease. In one case, a 69-year-old woman with early primary open-angle glaucoma and small Bjerrum scotomas, the eyes were removed for study immediately after her death from multiple myeloma.

The cases of secondary glaucoma all evidenced open filtration angles with no anterior or posterior synechias. The diagnostic data regarding the patients with secondary glaucoma are listed in Table 2. The average age of these patients was much lower than the average age of the patients with primary glaucoma. In many of the cases the original inciting cause (blunt injury, chorioretinitis, hemorrhage, and so forth) had subsided years before; but the glaucoma continued and progressed until enucleation was necessary.

Three patients with glaucoma of doubtful origin with Descemet's membrane extending over the filtration angle were examined but were not included in this study because it was felt that the profound degeneration found in the trabecular meshwork in these cases was not the subject of the present investigation.

Two simple experiments, which will be described later, were carried out on eyes from the Stanford Eye-Bank to help elucidate the meaning of some of the histologic variations noted.

Results

The study of sections of the trabecular meshwork in every plane reveals the nature and extent of the degenerative lesions much more clearly than the study of sections in the meridional plane alone. In most instances the tangential and transverse sections are particularly revealing but, in others, the meridional sections furnish information which cannot be obtained in any other way.

Striking pathologic changes are noted in the trabecular meshwork of eyes with primary open-angle glaucoma (figs. 1 to 9). These changes are degenerative in nature. They begin and are most marked on the external part of the trabecular meshwork or the inner wall of Schlemm's canal. The extent of the degeneration varies markedly in different areas of the limbus. In a given eye the meshwork may appear almost normal at the 2-o'clock position and show severe degeneration at the 4-o'clock position. In these respects (1) the presence of degenerative changes in the trabecular meshwork and

TABLE 1

LIST OF CASES: PRIMARY OPEN-ANGLE GLAUCOMA

No.	Source	Pertinent History	Age (yr.)	Tension (mm. Hg)	Other Eye
1 & 2	Stanford Eye Bank	Gradual blurring of vision. Occasional morning headaches. Tension frequently in mid-thirties, but usually well controlled with pilocarpine. Bilateral small Bjerrum scotomas. Vision 20/30 both eyes. Incipient cataracts. Died of multiple myeloma.	69	35	Primary open-angle glaucoma, both eyes
3	AFIP 499450 Right eye	Right eye began to lose vision 4 yr. prior to enucleation. No light perception.	69	40+	Chronic simple glaucoma
4	AFIP 499450 Left eye	Left eye began to fail 5 yr. prior to enucleation. Blind.	69		Chronic simple glaucoma
5	Stanford Eye Path. 56-10	Poor vision 1 yr. Painful 1 wk. Recent hyphemia. No light perception.		114	Glaucoma simplex; tension, 43
6	AFIP 686098	Blind painful left eye for 8 mo.	55	Stony hard	Chronic simple glaucoma controlled with miotics
7	AFIP 720864	Primary open angle glaucoma, 23 yr. duration. Pilocarpine 20 yr. Visual field constricted 18 yr. Tension elevated 5 yr. No vision.	76	100 (McLean)	Chronic simple glaucoma
8	AFIP 577292	Loss of vision 6 yr. Pain 4 wk.	?	60+	Primary open-angle glaucoma
9	AFIP 638508	Gradual loss of vision 13 yr. Optic nerve atrophic.	72	100	Blind of chronic glaucoma
10	AFIP 732016	Eye had been blind for 20 yr.	69	53	Chronic open-angle glaucoma; tension, 32
11	Stanford Eye Path.	Loss of vision 1 yr. No light perception.	84	70	Primary open-angle glaucoma; iridencleisis
12	AFIP 260484	Tension elevated in both eyes. Field constricted to 10°, pain 3 wk. Light perception.	56	60	Chronic glaucoma; enlargement of blind spot; tension, 40
13	AFIP 684933	One sister has glaucoma. Glaucoma; deep chamber; complete glaucomatous cupping. Branch occlusion of central retinal vein.	71	60	Small central cupping; tension, 22
14	AFIP 699279	No history of injury. Recent severe pain. Duration of blindness unknown. Deep cupping of disc. Open angles.	73	100	Vision, finger counting Macular scar? Tension, 23
15	AFIP 586616	No history of injury. Glaucoma many yr. Corneal ulcer and pain for 3 wk. Deep cupping of disc. Open angles.		65	Normal?
16	AFIP 270786	Pain for 1 wk. Absolute glaucoma. Complete cupping of disc. Open filtration angles.	78	90	Apparently normal at time affected eye was enucleated

TABLE 2

SECONDARY GLAUCOMA WITH OPEN FILTRATION ANGLES

No.	Cases	Diagnosis	History	Age (yr.)	Race	Other Eye	Tension (mm. Hg)
1	AFIP 724563	Glaucoma secondary to contusion.	Blunt injury 7 yr. prior to enucleation. Eye blind for 4 yr.; hard and painful for 2 wk.	37	N	Normal	Hard
2	AFIP 680536	Glaucoma secondary to trauma	Injury while hammering. Cataract and secondary glaucoma.	36	W	Normal	65
3	AFIP 636508	Glaucoma secondary cause and type undetermined	Corneal leukoma; keratitis; progressive loss of vision; light perception only.	35	N	Normal	Hard
4	AFIP 654466	Glaucoma secondary to old chorioretinitis; possible toxoplasmosis	Severe old chorioretinitis scarring; pain for 3 wk.	63	W	Normal	40
5	AFIP 543663	Glaucoma secondary to old endophthalmitis	Recurrent attacks of pain for 15 yr.; secondary cataract.	?	W	Normal	
6	AFIP 716059	Glaucoma secondary to injury and dislocated lens	Blind since injury. Enucleation because of endophthalmitis and sclerosing keratitis.	29	W		
7	AFIP 759801	Glaucoma secondary to hemangioma of the choroid (Sturge-Weber syndrome)	Blind painful left eye, glaucomatous since early childhood.	42	W	Normal	
8	AFIP 672943	Secondary glaucoma cataract	Senile cataract in other eye. Affected eye blind.	77	W	Normal tension	Hard
9	AFIP 697467	Aphakia, glaucoma	Onset of glaucoma after cataract extraction. Iridectomy 3 yr. later failed to control glaucoma. Eye blind.	89	W		Hard
10	666 Episcopal EENT Hosp. Wash., D. C. Contribution of Dr. Benjamin Reeves	Glaucoma secondary to central vein thrombosis	A fibrovascular membrane is not present in the iris. No synechias. Marked pigmentation of the iris due to hemorrhage and which stains for iron.	55	N	Normal	56
11	AFIP 502446	Glaucoma secondary to intraocular foreign body and siderosis bulbi.	Dislocated cataractous lens. Keratitis.	41	W	Normal	Stony Hard

(2) the site of these changes in the meshwork, the work of Teng, Paton, and Katzin[28] is confirmed. Degenerative changes in the collector channels were not as marked or as consistent as the trabecular changes.

Thickening of the trabecular beams and sclerosis of the trabeculae, as noted by Theobald and Kirk,[29] are seen in most of these eyes but are not invariably present. These changes are not marked in the least advanced cases.

The interlamellar stenosis reported by

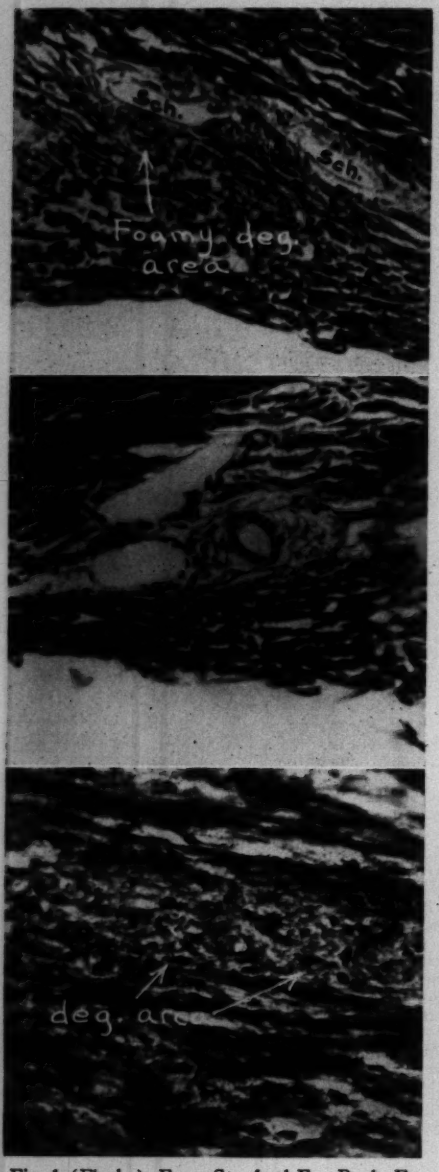

Fig. 1 (Flocks). From Stanford Eye-Bank. Eyes of 69-year-old white woman with primary open-angle glaucoma, both eyes. Tension 25 to 35 mm. Hg in both eyes with pilocarpine; small Bjerrum scotoma in each eye. Vision 20/30 both eyes; incipient cataracts. Patient died of multiple myeloma. (Top) Meridional section, with Masson's strain. Foamy degenerated appearance of outer part of meshwork. (Center) Meridional section, with Masson's stain. A blood vessel is present in the foamy area near Schlemm's canal. (Bottom) Tangential section; corneoscleral meshwork; Verhoeff's stain. A degenerated area is present.

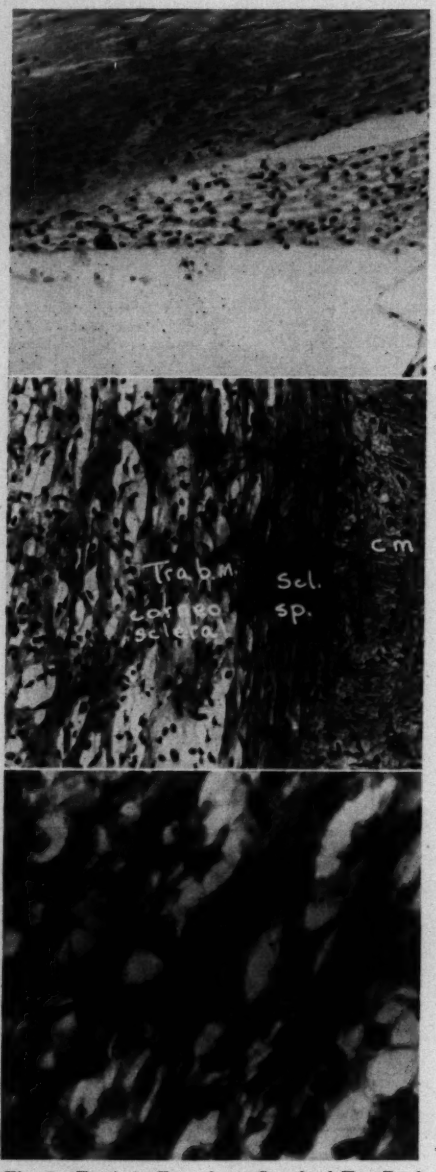

Fig. 2 (Flocks). Eyes from Stanford Eye-Bank. Normal meshwork for purposes of comparison. (Top) Meridional. (Center and bottom) Corneoscleral tangential sections. Note the plump, uniform quality of the trabecular nuclei.

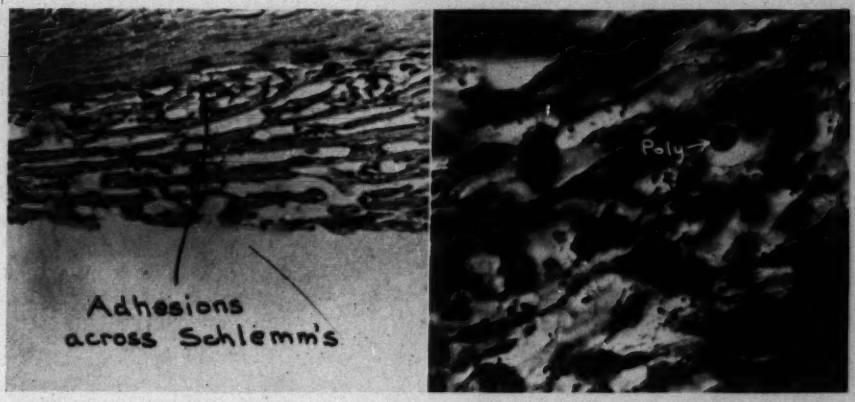

Fig. 3 (Flocks).—AFIP 577292. Primary open-angle glaucoma in both eyes. Loss of vision for six years. Tension, 60 mm. Hg. (Left) Meridional section. Schlemm's canal is virtually obliterated by adhesions across it. (Right) Tangential section near inner wall. Foamy granular appearance; increase in pigment; occasional inflammatory cell.

François and his colleagues[14] is frequently seen but is believed to be merely a change due to the immediate effect of increased intraocular pressure. The newly formed blood vessels in the region of Schlemm's canal which they report in one case of secondary glaucoma and call "rubeosis" of the trabeculae is found in this study in cases of primary and secondary glaucoma.

The pathologic changes seen in the trabecular meshwork and Schlemm's canal in these eyes with primary open-angle glau-

Fig. 4 (Flocks)—AFIP 720864. Eye of a patient 76 years of age. Primary open-angle glaucoma in both eyes of 23 years' duration; pilocarpine therapy for 20 years. Meridional section. Schlemm's canal is virtually obliterated by adhesions across it.

Fig. 5 (Flocks). AFIP 686098. Age of patient, 55 years. Primary open-angle glaucoma in both eyes. Tension stony hard. Oblique, almost transverse, section. Neovascularization of inner wall of Schlemm's canal.

coma are listed below. All of these changes were not noted in every eye but some were seen in each eye.

1. Narrowing and partial obliteration of Schlemm's canal.
2. A thickening and foamy appearance of the tissue just beneath Schlemm's canal (the external part of the trabeculae, the "pore area").
3. Frequently an increase in the number of cells on the inner wall. Most of the new cells are probably fibroblasts. Occasionally a few inflammatory cells are seen. In many instances with marked fibrosis, there is a decrease in the number of nuclei in the trabeculae. The cells seen in the meshwork are frequently less plump and less uniform in appearance.
4. Narrowing of the interlamellar spaces, as seen in routine section, with the nuclei assuming a flattened appearance.
5. Fibrosis and adhesions in Schlemm's canal.

Fig. 6 (Flocks). AFIP 638508. Age of patient, 72 years. Primary open-angle glaucoma in both eyes. Gradual loss of vision for 13 years. Marked degeneration and scarring of trabecular meshwork.

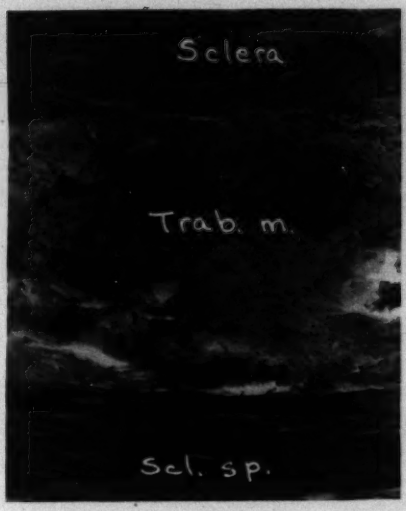

Fig. 7 (Flocks). AFIP 586616. Glaucoma of many years' duration. Open filtration angles. Cupped disc. Condition of other eye unknown. Dense scarring of trabecular meshwork.

6. Recanalization of Schlemm's canal by proliferation of endothelium along adhesive bands.
7. Vascularization of this area. Schlemm's canal is sometimes divided into small compartments which are indistinguishable from blood vessels and frequently contain blood.
8. Thickening of uveal and corneoscleral beams with an increase in collagen content in these beams.
9. Hyalinization of the meshwork, particularly the external part.
10. An increase in pigment having a characteristic distribution is usually seen. Pigment having the same distribution is seen in normal eyes of older individuals but is usually more marked in the glaucomatous eyes. The largest part of the pigment is contained in large melanophores or macrophages containing the granules, but some of it is located in the trabecular cells and some of it lies free. Most of it was located either in the uveal meshwork in large melanophores, or in the inner wall of Schlemm's canal, the bulk of the corneoscleral meshwork being comparatively free of pigment.

Some of the less marked changes are sometimes found in normal eyes but the more severe changes such as vascularization,

Fig. 8 (Flocks). AFIP 699279. Open-angle glaucoma. Tangential section. Large melanophores are seen in the uveal meshwork.

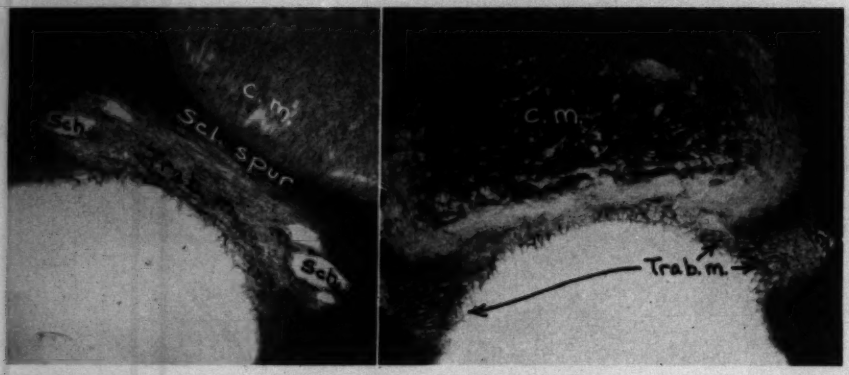

Fig. 9 (Flocks). (Left) AFIP 67294. Eye enucleated because of melanoma of choroid. Normal meshwork. Masson's stain. Curved tangential section for comparison with similar section (Right) AFIP 499450. Age of patient, 69 years. Primary open-angle glaucoma. Marked sclerosis of trabecular meshwork and ciliary body.

marked fibrosis, and hyalinization with complete loss of normal architecture (in tangential section) that are frequently seen in the eyes with primary open-angle glaucoma are never seen in normal eyes.

Secondary glaucoma with open filtration angles

The pathologic findings in the trabecular meshwork of the eyes with secondary glaucoma were quite surprising. Precisely the same types of degeneration that were found in primary open-angle glaucoma were seen in the filtration angles of these eyes (figs. 10 to 12). Although some of these individuals were quite young, sclerosis, degeneration, and increase in collagen were marked in almost every case. The meshworks were quite unlike those of normal eyes and every one of the pathologic changes that could be found in primary open-angle glaucoma was found in these eyes. The degeneration was, in general, just as extensive in these eyes as those with primary open-angle glaucoma. As occurred in the cases with primary open-angle glaucoma, some eyes had less extensive changes than others.

What is the meaning of these histologic alterations, some of which are found to be of small degree in eyes of normal older individuals and of much greater degree in eyes of patients with primary or secondary open-angle glaucoma?

The histologic changes may be divided into four groups:

1. Changes due to age.
2. The increase in pigment and its distribution.
3. Changes due to the immediate effects of increased intraocular pressure.
4. Changes due to the effects of prolonged increased intraocular pressure.

1. *Changes due to age*

In normal eyes of individuals of advanced age the trabecular beams become thicker and contain more collagen. This is in agreement with findings of Theobald,[29] Verhoeff,[30] Rones,[25] and others. Degenerative changes in the external trabeculae, thickening, and a foamy appearance of the area are sometimes present. The extensive changes such as vascularization, recanalization or obliteration of Schlemm's canal, marked fibrosis, and hyalinization are not found normally.

2. *The increase in pigment and its distribution*

The distribution of pigment in the meshwork is evidence that the smallest openings or chief resistance to the passage of pigment from the anterior chamber to the outside of

Fig. 10 (Flocks). AFIP 636508. 35-year-old Negro with unilateral secondary glaucoma with open filtration angles. Corneal leukoma. Progressive loss of vision. No synechias. Optic disc cupped. Other eye normal.

(Top) Meridional section. The meshwork is compressed and appears to be pushed anteriorly, obliterating Schlemm's canal. The pigment is heaviest near the inner wall of Schlemm's canal. Compare with Figure 13 and Figure 14.

(Center) Meridional section opposite angle. New adhesions are forming across Schlemm's canal. An increased number of cells is present in and near Schlemm's canal.

(Bottom) Tangential section. Degenerated area of corneoscleral meshwork containing acute and chronic inflammatory cells.

the eye through the filtration angle are in the region of the inner wall of Schlemm's canal, the "pore area" (fig. 13). The larger cells and clumps of pigment are stopped at the uveal meshwork, but most of the pigment collects near the inner wall of Schlemm's canal, in the "pore area," the great bulk of the corneoscleral meshwork being left relatively free of pigment. It is evident that the pigment has difficulty in getting through this area because of the relatively smaller openings in the meshwork.

By injecting diluted India ink, the same distribution of ink has been produced in our laboratory in eyes from the eye-bank, the larger clumps of pigment being caught in the uveal meshwork and the bulk of it being stopped near the inner wall of the canal. Very little pigment or India ink remains in the vessels distal to Schlemm's canal, for it is clear that if the particles are small enough to get through into Schlemm's canal, they have relatively clear sailing from then on. This is not surprising, for we know that the caliber of the vessels between the outer surface of the eyeball and Schlemm's canal is large enough to allow red blood cells to pass into Schlemm's canal, but that the red cells cannot pass through the wall of Schlemm's canal into the anterior chamber.

In addition, the openings of the uveal and corneoscleral meshworks as seen in tangential section are larger than a red blood cell until the region of the inner wall of Schlemm's canal is reached.[9] Here the openings are smaller and hard to see. Huggert[20] and François and his co-workers[13] in separate perfusion experiments demonstrated that the size of the smallest openings, the "pore size," is about two or three microns.

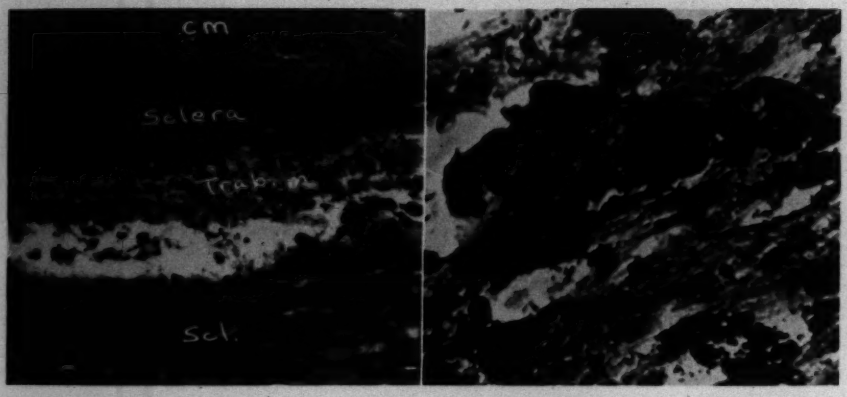

Fig. 11 (Flocks). AFIP 742563. 37-year-old Negro. Glaucoma secondary to contusion. Blunt injury seven years prior to enucleation. Blind for four years. Painful eye for two weeks. Other eye normal. (Left) Tangential section. There is marked scarring and loss of normal architecture of the corneoscleral meshwork. Pigment deposits are increased. (Right) Tangential section; high power. The number of trabecular cells is decreased.

The fact that the amount of pigment increases moderately with age but increased greatly in primary and secondary open-angle glaucoma suggests that the daily variations in pressure, and particularly the increase in pressure, dislodges pigment and pigment-bearing cells from the iris, which are then caught and stopped in the trabecular meshwork.

The increase in pigment in the meshwork

Fig. 12 (Flocks). AFIP 697467. Secondary glaucoma. Onset of unilateral glaucoma three months after cataract extraction. Iridectomy three years later failed to control glaucoma. Meridional section. Canal obliterated by scar tissue containing an increased number of nuclei. Neovascularization of Schlemm's canal.

Fig. 13 (Flocks). Eye from eye-bank. Oblique section. India ink solution made to flow through the eye by injection into the anterior chamber of eye-bank eyes tends to collect near the inner wall of Schlemm's canal, indicating that the openings in the meshwork are smallest here. Compare with Figure 10 (top).

is certainly not due to aging alone. We have an infant's eye with congenital glaucoma in which tangential sections of the meshwork show a marked increase in the amount of pigment in the trabeculae.

3. *Changes due to the immediate effects of increased intraocular pressure*

When the pressure in the anterior chamber of the eye is increased, the trabecular meshwork is pushed forward (or stretched) compressing the trabecular lamellae and narrowing Schlemm's canal. These findings were present in almost every one of the glaucomatous eyes. This forward displacement (or stretching) and compression of the meshwork are due simply to increased pressure. That was shown in an experiment suggested to me by Dr. Otto Barkan. Desiring to see the histologic appearance of the trabeculae when the anterior chamber is deepened, he suggested that a warm solution of gelatin be injected into the anterior chamber of an eye from the eye-bank under a slight pressure. Immediately after the gelatin is injected, it cools and solidifies in the anterior chamber, keeping it deep and under increased pressure. When the eye is fixed and examined histologically the trabecular lamellae are seen to be compressed and pushed forward (or stretched), narrowing Schlemm's canal, just as is found in the majority of glaucomatous eyes (fig. 14).

The forward displacement of the trabeculae with narrowing of Schlemm's canal found in glaucomatous eyes is further evidence that the site of obstruction to aqueous outflow is in the trabeculae near the inner wall of Schlemm's canal. If the chief obstruction to outflow were in the sclera, distal to Schlemm's canal, one would expect Schlemm's canal to be dilated instead of narrowed, but this is never the case. Indeed, the histologic, experimental, and mathematical evidence that the chief obstruction to aqueous outflow is on the proximal rather than distal side of Schlemm's canal is now overwhelming,[2, 10, 16, 23] but will not be discussed in detail at this time.

The nuclei of the trabecular cells appear elongated and compressed in almost all of the glaucomatous eyes but whether this is due to the immediate or to the prolonged effect of increased intraocular pressure is not yet certain.

4. *Changes due to the prolonged effects of increased intraocular pressure*

Let us first consider the cases of secondary glaucoma with open filtration angles. These cases have diverse causative factors. The original increase in intraocular pressure in some cases may have been produced by blockage of the outflow channels by hemorrhage, inflammation, pigment, or intraocular foreign body; or for mechanical reasons such as in intumescent cataract. The tension in many of these cases subsided for a time after the initial bout of glaucoma and then gradually rose to the point where enuclea-

Fig. 14 (Flocks). Eye from eye-bank. Pressure in anterior chamber increased by the injection of gelatin. The trabecular meshwork is compressed, narrowing Schlemm's canal.

tion was done. In all of these cases essentially the same lesions of the trabeculae are seen, but they are in various stages of development in different parts of the meshwork and in different eyes.

First there is an edema or foamy appearance of the inner wall of Schlemm's canal, with an increased number of nuclei in this area. With the canal narrowed and this degenerative process going on in the inner wall, adhesions develop between the inner and outer wall of Schlemm's canal, dividing the canal into compartments with what appears to be new blood vessels or attempts at recanalization of the canal. Later the number of nuclei in the external trabeculae decreases as fibrosis, hyalinization, and thickening of the trabecular beams are seen.

The only two things that all of these cases have in common are increased intraocular pressure and degenerative changes in the filtration angle. It is quite clear that these degenerative changes are a result of the prolonged increased intraocular pressure.

In primary open-angle glaucoma, precisely the same degenerative changes are seen in the trabecular meshwork; here, too, these changes must be due to an intraocular pressure high enough to cause trabecular damage.

Pathogenesis of primary open-angle glaucoma

The degenerative changes seen in the external trabeculae and Schlemm's canal in cases of primary open-angle glaucoma are evidently the cause of the reduced facility of outflow. We have seen that this type of trabecular damage is produced in cases of secondary glaucoma with open angles by prolonged increased intraocular pressure.

In eyes destined to have primary open-angle glaucoma, the tension at first is not high. What, then, causes, the original trabecular change? Pathologic and clinical evidence combine to suggest the answer to this question.

One of the most characteristic findings of primary open-angle glaucoma is a failure of the pressure-regulating mechanism. The diurnal variations in intraocular pressure are believed to be due to variations in the production of aqueous. One of the earliest manifestations of primary open-angle glaucoma is an increase in the diurnal variations of the intraocular pressure. This is taken as evidence that the pressure-regulating mechanism is becoming less able to control the changes in intraocular pressure caused by

variations in aqueous outflow. Swanljung and Blodi,[27] Kronfeld,[22] and deRoetth[7, 8] have pointed out that in the glaucomatous eye the facility of outflow is relatively rigid, changing very little in response to provocative testing or other stimulus.

There is good evidence that the trabecular meshwork is a pressure-regulating mechanism.[2, 10, 11, 31] The degenerative changes seen in the trabeculae of eyes with open-angle glaucoma can explain the failure of the pressure-regulating mechanism in this disease.

There is a large familiar factor in the incidence of primary open-angle glaucoma. It has been estimated that in 13 to 25 percent of the cases of primary open-angle glaucoma, some member of the patient's family has the disease.[18, 21] Further studies show that in families with some member having the disease there is a higher percentage than usual of individuals with a "high normal" tension.[21]

Bennett[5] has found that the life expectancy of patients with glaucoma is normal. If early generalized sclerosis or degeneration of connective tissue (which has been suggested as a possible cause of primary open-angle glaucoma) occurred in these people, we would expect that their life span would be shortened.

We have seen that some trabecular change occurs in normal eyes of some aged individuals. We know that as the age increases in normal individuals, the tension gradually rises, and this gradual increase in tension with age could conceivably be due to slight damage to the pressure-regulating mechanism.

We know that prolonged increased intraocular pressure will damage the optic nerve, the ciliary body, and now, in cases of secondary glaucoma, the trabecular meshwork.

In primary open-angle glaucoma, the individual must have vulnerable trabeculae, possibly inherited, which are injured by a level of pressure lower than that of other individuals and lower than that which would ordinarily damage the optic nerve or ciliary body. For example, if an individual had a trabecular meshwork which would be injured whenever the tension rose to 24 mm. Hg, and if at the age of 38 years the tension had risen (with the normal decrease in facility of outflow with age[32]) to the point where it reached 24 mm. Hg daily at the peak of the diurnal curve, trabecular damage as described earlier would occur daily, causing the facility of outflow to decrease steadily. The optic nerve might not begin to suffer until the tension was reaching 32 mm. Hg, which might be years later. The glaucoma had begun, however, when the tension was reaching 24 mm. Hg, a level at which progressive degeneration of the trabecular meshwork was assured.

It is also possible that the predisposed trabeculae, as they grow older, "wear out" like joints do in a patient with hypertrophic arthritis, so that they are no longer able to withstand the normal pressures that they formerly could, and are damaged by these normal pressures.

In brief, according to this theory, primary open-angle glaucoma is caused by the presence of vulnerable trabeculae or a pressure-regulating mechanism which is damaged by levels of intraocular pressure which ordinarily do not damage the trabeculae or optic nerve. This injury interferes with aqueous drainage and promotes an increase in intraocular pressure which further damages the trabecular meshwork.

Clinical implications

This new concept of the pathogenesis of open-angle glaucoma is attractive because with it one can readily explain many of the puzzling clinical findings of glaucoma.

a. *Juvenile glaucoma* can be thought of as occurring in a patient with very susceptible trabeculae so that the critical tension is very low and is reached at an early age, producing primary open-angle glaucoma very early.

b. *Combined glaucoma.* Although it is undoubtedly true that some patients with combined glaucoma (patients with both a narrow-angle glaucoma and a reduced facility

of outflow when the angles are open) start out with primary open-angle glaucoma and later develop an enlarged lens and a superimposed narrow-angle mechanism, this new concept suggests that in some cases the reverse may be true. That is, in some cases of narrow-angle glaucoma, the periodic elevations of tension may cause enough trabecular damage to cause a reduced facility of outflow even though no synechias are present and the angles are open.

c. *Low tension glaucoma* can be thought of as occurring in an individual in whom the optic nerve and retina are susceptible to damage by relatively low levels of intraocular pressure but the trabecular meshwork, the pressure-regulating mechanism, is normally resistant and is not damaged excessively. Consequently, there is no progressive decrease in aqueous outflow even though the optic nerve undergoes degeneration.

d. *Secondary glaucoma.* As described earlier, cases of secondary glaucoma in which the original cause of the rise in tension has disappeared but the glaucoma remains or reappears even though no synechias are present can be explained in the same manner; namely, that trabecular damage occurred during the original period of elevated tension.

e. *Management of early primary open-angle glaucoma.* In primary glaucoma, if pressure is what caused the progressive degeneration of the meshwork and the progressive decrease in facility of outflow, then early diagnosis and treatment is of the utmost importance. It is during the earliest period of the disease, when the pressure has not reached a level that we ordinarily think of as pathologic, and before optic nerve changes have occurred, when the greatest opportunity to halt the process of trabecular damage is presented. If our conception of open-angle glaucoma is true, then our aim in treatment must be to lower the intraocular pressure *enough* to avoid further trabecular damage. This desired tension may be far below that necessary to prevent loss of field. To illustrate, a patient with early open-angle glaucoma may, with miotics, have his tension lowered to 28 mm. Hg. At this level, over a period of a year or two, no progression of the field defect may occur. But if trabecular damage is occurring at this level of intraocular pressure, the pressure will eventually rise. If the tension could have been lowered sufficiently, perhaps to 22 mm. Hg, it is possible that no further trabecular damage would occur, and the glaucoma would never progress.

Recently Goedbloed[15] in a very interesting study has pointed out that the prognosis and response to treatment of primary open-angle glaucoma is much better if the diagnosis is made very early before the appearance of any field defect, and Smillie and his colleagues[26] and Brav and Kirber[6] have made similar statements.

Fralick[12] has stated that families of patients with primary open-angle glaucoma are a fertile field for glaucoma case finding and has suggested periodic glaucoma workups for the relatives of patients with the disease after the age of 40 years. If failure of the pressure-regulating mechanism of these individuals becomes manifest, treatment with miotics should be begun early with, according to our new concept, good expectations for success in treatment.

Becker and Christensen[3] and Hildreth and Becker[19] have also recently stressed the clinical importance of early determination of primary open-angle glaucoma and have delineated new techniques of early case findings.

Confirmation

The pathologic changes in cases of primary and secondary glaucoma can be readily seen. The theory of the pathogenesis of primary open-angle glaucoma, however, must await final proof. We are at present attempting to see whether prolonged elevation of intraocular pressure in animals will produce these degenerative lesions of the meshwork. We must make further careful histologic studies of the filtration angles of cases of primary and secondary glaucoma. Finally,

the clinical implications of the theory must be tested in the crucible of clinical experience.

Summary

Histologic study of the trabecular meshwork of 16 cases of primary open-angle glaucoma and 11 cases of secondary glaucoma with open filtration angles in meridional, transverse, and tangential sections reveal marked degenerative changes in the meshwork and Schlemm's canal, beginning in the external portion of the trabecular meshwork. The location and character of the degenerative changes indicate that they are responsible for the decreased facility of aqueous outflow characteristic of primary open-angle glaucoma. It is apparent from the congruity of the histologic alterations in primary glaucoma and in secondary glaucoma with open filtration angles that these degenerative changes are caused by prolonged increased intraocular pressure.

A new theory of the pathogenesis of primary open-angle glaucoma is proposed. It is suggested that persons destined to have primary open-angle glaucoma have relatively vulnerable trabeculae which are injured by levels of intraocular pressure which ordinarily do not damage the trabeculae or optic nerve. When the curve of intraocular pressure, which normally slowly rises with advancing age, reaches the point where enough trabecular degeneration has occurred to cause insufficiency of the pressure-regulating mechanism, progressive glaucoma is assured unless treatment is instituted. This theory implies that the aim of therapy in early glaucoma should be to lower the intraocular pressure sufficiently to prevent further trabecular damage, rather than lowering it enough merely to prevent damage to the optic nerve; for if the trabeculae continue to be injured, the pressure will inevitably rise.

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References

1. Ashton, N., Brini, A., and Smith, R.: Anatomical studies of the trabecular meshwork of the normal human eye. Brit. J. Ophth., **40**:257-282 (Feb.) 1956.
2. Bárány, E. H.: The physiology and pathology of the filtering angle. In Glaucoma: A Symposium. (William Stewart Duke-Elder, ed.) Springfield, Ill., Thomas, 1955, pp. 91-104.
3. Becker, B., and Christensen, R. E.: Water-drinking and tonography in the diagnosis of glaucoma. Arch. Ophth., **56**:321-326 (Sept.) 1956.
4. Becker, B., and Constant, M. A.: The facility of aqueous outflow. Arch. Ophth., **55**:305-312 (Mar.) 1956.
5. Bennett, G.: Mortality rates in glaucomatous subjects. Arch. Ophth., **54**:637-640 (Nov.) 1955.
6. Brav, S. S., and Kirber, H. P.: Mass screening for glaucoma. J.A.M.A., **147**:1127-1128 (Nov.) 1951.
7. de Roetth, A., Jr.: Effect of changes in osmotic pressure of blood on aqueous humor dynamics. Arch. Ophth., **52**:571-582 (Oct.) 1954.
8. de Roetth, A., Jr., and Schwartz, H.: Aqueous humor dynamics in glaucoma. Arch. Ophth., **55**:755-764 (June) 1956.
9. Flocks, M.: The anatomy of the trabecular meshwork as seen in tangential section. Arch. Ophth., **56**: 708-718 (Nov.) 1956.
10. ———: Unpublished data.
11. Flocks, M., and Zweng, H. C.: Studies on the mode of action of pilocarpine on aqueous outflow. Am. J. Ophth., **44**:380-388 (Nov., pt. II) 1957.
12. Fralick, F. B.: Glaucoma detection and treatment. Sight-Saving Rev., **25**:194-199 (Winter) 1955.
13. François, J., Neetens, A., and Collette, J. M.: Microradiographic study of the inner wall of Schlemm's canal. Am. J. Ophth., **40**:491-500 (Oct.) 1955.
14. François, J., Rabaey, M., and Neetens, A.: Perfusion in vitro of 10 glaucomatous eyes. Arch. Ophth., **55**:488-502 (Apr.) 1956.
15. Goedbloed, J.: The prognosis of simple glaucoma. Ophthalmologica, **132**:181-184 (Sept.) 1956.
16. Goldmann, H.: An analysis of primary glaucoma. Tr. Ophth. Soc. U. Kingdom, **69**:455-476, 1949.
17. Grant, W. M., and Trotter, R. R.: Tonographic measurements in enucleated eyes. Arch. Ophth., **53**:191-200 (Feb.) 1955.
18. Havener, W. H.: Chronic simple glaucoma: hereditary aspects. Am. J. Ophth., **40**:828-831 (Dec.) 1955.

19. Hildreth, H. R., and Becker, B.: Routine tonometry. Am. J. Ophth., **43**:21-23 (Jan.) 1957.
20. Huggert, A.: Pore size in filtration of eye. Acta Ophth., **33**:271-284, 1955.
21. Kellerman, L., and Posner, A.: The value of heredity in the detection and study of glaucoma. Am. J. Ophth., **40**:681-685 (Nov., pt. I) 1955.
22. Kronfeld, P. C.: Correlations between low facility of outflow and other characteristics of chronic simple glaucoma. Acta, XVII Internat. Cong. Ophth. (1954), **3**:1553-1556, 1955.
23. Linnér, E.: The outflow pressure in normal and glaucomatous eyes. Acta Ophth., **33**:101-116, 1955.
24. Maggiore, L.: Quoted by Theobald and Kirk.[29]
25. Rones, B.: Senile changes and degenerations of the human eye. Am. J. Ophth., **21**:239-255 (Mar.) 1938.
26. Smillie, J. W., Roth, F. D., Blum, F., and Gates, L. K.: Glaucoma detection in a general hospital. Am. J. Ophth., **44**:20-23 (July) 1957.
27. Swanljung, H., and Blodi, F. C.: Tonography in some provocative tests for glaucoma. Am. J. Ophth., **41**:187-197 (Feb.) 1956.
28. Teng, C. C., Paton, R. T., and Katzin, H. M.: Primary degeneration in the vicinity of the chamber angle. Am. J. Ophth., **40**:619-631 (Nov., pt. I) 1955.
29. Theobald, G. D., and Kirk, H. Q.: Aqueous pathways in some cases of glaucoma. Am. J. Ophth., **41**:11-21 (Jan.) 1956.
30. Verhoeff, F. H.: Quoted by Theobald, and Kirk.[29]
31. Weekers, R., Delmarcelle, Y., Prijot, E., and Lavergne, G.: Modification of outflow facility. Am. J. Ophth., **43**:899-907 (June) 1957.
32. Weekers R., Watillon, M., and de Rudder, M.: Experimental and clinical investigations into the resistance to outflow of aqueous humour in normal subjects. Brit. J. Ophth., **40**:225-233, 1956.

TWO NEW CORTICOSTEROID PREPARATIONS*

Used in the treatment of ocular disorders

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Recently two topical formulations were made available which were developed to take advantage of the reported interaction and possible synergism of other compounds with lower doses of the predni-steroids in treating ocular disorders.[1-14] One of the formulations contains an antibiotic,† the other does not.‡ This report presents clinical experiences with these preparations in 101 patients presenting a variety of anterior segment disorders.

Clinical rationale

One formulation contains prednisolone alcohol 0.2 percent, neomycin sulfate 0.5 percent, and phenylephrine HCl 0.12 percent in a lubricating solution; the other contains prednisolone acetate 0.12 percent, phenylephrine HCl 0.12 percent, and methylcellulose 0.12 percent in a buffered aqueous suspension.

Prednisolone alcohol has been reported to be clinically effective in suspension form in an optimum concentration of 0.25 percent.[1] Another study indicated that 0.1 percent and 0.2 percent prednisolone alcohol solutions compare favorably in therapeutic effect with suspensions.[9] Consequently, these concentrations appeared desirable for evaluation.

Neomycin sulfate[17-20] can present a veritable mountain of references to support its claim of being a preferred topical antibiotic; topically effective with no systemic absorption or effect, minimum of sensitization, and lack of development of resistant micro-organisms. Neomycin is indicated in the majority of superficial eye infections—a very small proportion are caused by organisms not controllable with neomycin. The most common bacteria which cause conjunctival infections are the Morax-Axenfeld diplobacillus, the

* From the New York Hospital-Cornell Medical Center and the L. Margolyes League.
† Available as Predmycin Ophthalmic Solution, Allergan Corporation.
‡ Available as Prednefrin Ophthalmic Suspension, Allergan Corporation.

staphylococcus, the pneumococcus, Koch-Weeks bacillus, and streptococcus.[21] Neomycin is effective against this group as well as against Proteus vulgaris.[19,20]

"Neomycin sulfate exhibits activity against a variety of gram positive and gram negative bacteria.[23] In the former group it appears to be more effective against staphylococci than streptococci. It has a wider range than bacitracin, penicillin, or streptomycin, and it is sometimes effective against pseudomonas and proteus infections . . ." However, it is suggested that one should not rely upon neomycin in pseudomonal infection of the cornea. Here polymyxin is indicated. Vogel, et al.,[10] reported neomycin to be effective intraocularly in 0.5-percent concentrations.

Phenylephrine HCl has been reported to yield favorable results in allergic inflammations when combined with a steroid.[11-14] Other studies have indicated a synergistic relationship between the decongestant and hydrocortisone.[11,14] It has also been proposed that phenylephrine's decongestant action helps localize the corticoid in the tissue. In addition, phenylephrine HCl offers the advantage of an immediate paling agent.[7] Phenylephrine is probably the safest of the commonly used sympathomimetic drugs; especially in the low concentrations employed here.[15,16]

These topical preparations are indicated in those inflammatory, allergic, and infectious conditions which involve the lids, external eye, and anterior uvea. In anterior uveitis (iritis and iridocyclitis) topical steroid therapy is effective in approximately 60 percent or more cases if treated early in their course.[1,2,4] It is contraindicated in all cases of herpes simplex infections of the cornea (dendritic keratitis) and in certain exanthemas (chickenpox and smallpox) and fungus infections when these involve the eye. Fortunately, corneal exanthemas and fungal infections are rare in this country.

When a keratitis cannot be classified, steroid therapy should be withheld until dendritic keratitis can be diagnosed or ruled out. If one does prescribe steroids in a keratitis of dubious etiology, the patient should be seen daily for the first several days—if only briefly—in order to make certain that he is not developing a dendritic keratitis. In keratitis of dubious etiology, one should always test the corneal sensitivity before prescribing topical steroids. Herpes simplex characteristically causes corneal anesthesia.

The chief aim in the use of these prednisolone-phenylephrine-neomycin and prednisolone-phenylephrine-methylcellulose preparations topically is to achieve a maximal concentration of the steroid (and antibiotic) at the site of disease. To that end during the initial period of treatment, and especially during the severe phase of the disease, the drops should be instilled every half hour or every hour while the patient is awake. When the disease is controlled, the frequency of dosage can be decreased gradually. There is no systemic absorption from topically applied steroids.

A uevitis which does not respond to topical steroids within 48 to 72 hours should definitely be treated with added systemic steroids, as well as any specific medication deemed necessary. When infection is present or suspected, the steroid-phenylephrine-neomycin formulation is indicated, since an antimicrobial is mandatory in the presence of bacterial infection.

When a scaly blepharitis is to be treated, one must first manually remove the scales in order that the medication be given direct access to the lesions. If this simple precaution is taken, a higher degree of success will result from the topical therapy.

Postoperative or other granulation tissue—such as a retention granuloma or suture reaction after strabismus surgery—is a definite indication for one of these two preparations. The granulation tissue often will literally melt away. Reactions to suture materials also respond dramatically. When a severe postoperative reaction follows intraocular surgery, these topical preparations are frequently beneficial.

TABLE 1
CLINICAL RESULTS

Condition	Number of Patients	Frequency and Duration	Success	Failure	Comments
Acute catarrhal conjunctivitis	46	qid 5–11 days	46	0	Excellent results; one patient required silver nitrate
Chronic conjunctivitis	11	qid 12–21 days	9	2	2 cleared after Staph. toxoid
Chronic lid allergy	4	qid 18–35 days	4	0	Very good results
Episcleritis	3	qid 6–21 days	3	0	Excellent results
Chronic anterior uveitis	4	qid 49–80 days	4	0	Excellent results; also required systemic steroids
Chronic generalized uveitis	4	6 id 120 days	4	0	good control; also required systemic steroids
Acute anterior uveitis	6	6 id 14–30 days	5	1	One case required systemic therapy
Recurrent iritis	2	6–8 id 12–18 days	2	0	Good control; one also required systemic steroids
Severe reaction to chalazion surgery	3	6 id 4 days	3	0	Excellent results
Postsurgical retention granuloma	2	qid 2 months	2	0	Excellent results
Acute meibomitis	8	q2 hours. 4–6 days	6	2	Two cases not aborted; required surgery
Subacute meibomitis	3	6 id 3–5 days	0	3	All required surgery but lid reaction was reduced
Blepharitis	5	4–6 id 14–30 days	5	0	Excellent results
Totals	101		93	8	

CLINICAL RESULTS

Table 1 indicates the specific types of conditions treated, as well as the responses obtained.

SUMMARY

Two new formulations—(1) a prednisolone-phenylephrine-neomycin combination, (2) a prednisolone-phenylephrine-methylcellulose preparation—have been utilized clinically in a wide variety of superficial and anterior segment diseases with excellent results. They are especially indicated in inflammatory and allergic states, as well as in the diminution of granulation tissue masses.

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REFERENCES

1. Gordon, D. M.: Am. J. Ophth., **41**:593, 1956.
2. King, J. H., Jr., and Weimer, J. R.: Arch. Ophth., **54**:46, 1955.
3. King, J. H., Jr., Passmore, J. W., Skeehan, R. A., and Weimer, J. R.: Tr. Am. Acad. Ophth., **59**: 759, 1955.
4. Hogan, M. J., Thygeson, P., and Kimura, S.: Tr. Am. Acad. Ophth., **59**:777, 1955.
5. O'Rourke, J. F., Iser, G., and Ryan, R. W.: Arch. Ophth., **55**:323, 1956.
6. Duke-Elder, S., and Goldsmith, A. J. B.: Survey Ophth., **1**:172, 1956.
7. Tuft, L., and Neish, D. D., Jr.: J.A.M.A., **167**:60 (May) 1958.
8. Larson, G. A., and Fercho, C. K.: Am. J. Ophth., **44**:260, 1957.
9. Lippmann, O.: Arch. Ophth., **57**:339 1957.
10. Vogel, A. W., Leopold, I. H., and Nichols, A.: Am. J. Ophth., **34**:1357, 1951.

11. Silcox, L. W.: Arch. Otol., **60**:431, 1954.
12. Pennypacker, C. S.: J. Allergy, **25**:513, 1954.
13. Tuft, H. S.: Ann. Allergy, **12**:687, 1954.
14. Poetsch, C. E.: United States Patent No. 2,801,202, 1957.
15. Heath, P., and Geita, C. W.: Arch. Ophth., **41**:172 (Feb.) 1949.
16. Drill, V. A.: Pharmacology in Medicine. New York, McGraw-Hill, chap. 26, p. 12.
17. Leopold, I. H.: Tr. Am. Acad. Ophth., **58**:809 (Nov.-Dec.) 1954.
18. Gordon, D. M.: Survey Ophth., **3**:107 (Apr.) 1958.
19. Hallett, J. W., Wolkowicz, M. I., and Leopold, I. H.: Am. J. Ophth., **41**:850 (May) 1956.
20. Rhodes, R. E., and Ferlauto, R. J.: Antibiotics Annual 1953-54. Medical Encyclopedic Inc., Philadelphia, Blakiston.
21. Berens, C., and Siegel, E.: Encyclopedia of the Eye. Philadelphia, Lippincott, 1951, chap. 49, p. 8.
22. Welch, H.: Principles and Practice of Antibiotic Therapy, Medical Encyclopedia, Inc., Philadelphia, Blakiston, pp. 158-161.
23. Stormont, R. T. (editor): New and Nonofficial Remedies. Philadelphia, Lippincott, 1957, pp. 118-120.

MANAGEMENT OF TRAUMATIC LATERAL RECTUS PARALYSIS*

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It is the purpose of this paper to discuss certain aspects of the management of uncomplicated traumatic lateral rectus paralysis. This will include (1) methods of preoperative evaluation, (2) choice of surgical procedure, and (3) timing of surgery.

LITERATURE

Surgical treatment of lateral rectus paralysis has been a challenge since Hummelsheim[1] first reported in 1907 on vertical rectus tendon transplants as a means of obtaining horizontal rotation. His monkey experiments, interestingly enough, involved the excision of the anterior portion of the medial rectus and the nasal transplantation of the vertical recti to gain adduction.

Hummelsheim[2] is properly credited with performing the first clinical vertical rectus tendon transplant for lateral rectus paralysis. O'Connor[3,4] published numerous papers on the success of the tendon transplant operation and was for years one of its strong advocates. Many authors have added reports of cases and modifications of technique, and one of the recent series is that of Berens and Girard.[5] Much less has been written as to the specific indications for surgery or the choice of procedure.

Hill,[6] in 1955, presented a paper before this society on the nasal transplantation of the vertical recti to gain adduction, thus duplicating clinically what Hummelsheim actually did on the monkey in 1908. In discussion of this paper additional cases were cited by Berens,[7] McLean,[8] and Braley.[9] Another report has been contributed this year by Girard and McNeely.[10]

In spite of the clinical evidence that the Hummelsheim operation can alter horizontal rotation, there continues to be some controversy over the effectiveness or even the wisdom of vertical rectus tendon splitting operations. Comparison of results by different methods is understandably difficult because of the variable character of the individual case and the relatively small numbers operated by any one surgeon. To mention only two factors, there may be a complete paralysis or only a partial paresis;

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again, there may be a normal opposing medial rectus or a contracture of the antagonist, preventing even forceps rotation into the temporal field. Traumatic sixth nerve paralyses are particularly prone to develop a contracture of the medial rectus.

Scobee[11] believed that little more was gained by vertical rectus tendon transplants than by surgery on the medial and lateral rectus alone, claiming that much of the effect was due to recession of the opposing medial rectus. He recommended surgery on the primary horizontal rotators as a preliminary operation before any Hummelsheim type procedure, and insisted on an adequate interval to evaluate these results before proceeding with tendon transplants. Spaeth[12] and Krewson [13] have both favored recession of the medial rectus as the initial procedure. On the other hand, Hill reported a sixth nerve paralysis operated by this two-stage method that gained no abduction from horizontal surgery alone, but did attain satisfactory temporal rotation after the vertical tendon transplants. O'Connor reversed this routine and performed as the initial operation the transplantations from the vertical recti and reported definite abduction from this procedure alone.

Experimental

In 1956, I operated on a series of nine monkeys in an attempt to evaluate the effectiveness of different techniques of tendon transplants. Because of the recent scarcity of rhesus monkeys the original objective had to be abandoned before significant comparisons were available. If added confirmation of Hummelsheim's original experiment were needed, however, it was shown in these monkeys that vertical rectus split tendon transplants can by themselves produce abduction when transplanted to the lateral rectus stump. The efficiency of these transplants in favoring abduction was variable but in each case it was enhanced by a subsequent recession of the medial rectus.

In the process of studying the relationship between primary and secondary horizontal rotators, in 1954 I[14] performed a variety of whole tendon transplants. Particularly interesting was the transplant to the insertions of the primary horizontal rotators which were themselves left undisturbed. When the full tendons of the vertical recti were transplanted to the insertion of the intact medial rectus, no imbalance was produced. However, when the full tendons were transplanted to the insertion of the intact lateral rectus, there was gross divergence. This suggested that the augmentation of the secondary adductor function of the vertical recti obtained by nasal transplants was apparently not sufficient to upset the balance. When the alignment of the vertical recti was shifted well temporal to the center of rotation, however, apparently two things happened: the secondary adduction by these muscles was eliminated and the vertical recti were converted into an additional pair of secondary abductors.

It is my impression from post-mortem dissections on monkeys, that in the Hummelsheim operation the tendon slips frequently adhere to the sclera along the line of the transplant, giving the effect of elongated insertion of the vertical recti extending to the extremities of the lateral rectus stump. The mechanism whereby these transplants are effective has been nicely explained by Hildreth,[15] who has shown that the vertical recti may become abductors by a shift in the line of action of the main body of the muscle temporal to the center of rotation. This is in accord with the concept of co-contraction of the vertical recti as has been emphasized by Adler.[16]

Report of cases

The following two cases of traumatic sixth nerve paralysis are presented because of their bearing on the problem of contracture. In each instance a Hummelsheim procedure was considered but in neither patient was the tendon transplantation completed for the reasons indicated.

CASE 1

A 58-year-old white woman sustained a head injury in an auto accident in March, 1951. She had noted crossing of the right eye and constant double vision in all fields since the accident. Repeatedly she had been advised to "wait and see" whether function would not recover.

When examined on February 23, 1953, corrected visual acuity was 20/20 in each eye. There was marked convergence of the right eye with inability to abduct to within 25 degrees of fixation. She could fuse grossly in the primary position with 120 diopters base-out prism. The duction test under anesthesia revealed that it was not possible forcibly to rotate the globe out of the adducted position. Prior to ductions a Hummelsheim procedure had been contemplated but the medial rectus was found at surgery to be tense and fibrotic. Because of the shortened muscle considerable difficulty was experienced in exposing the tendon. A four-mm. recession of the medial rectus was accomplished and this was combined with an eight-mm. resection of the paralyzed lateral rectus.

Postoperative alignment was good in the primary position and there was fair adduction but no abduction. On the troposcope there was second-grade fusion on macular targets and 20 diopters convergence amplitude. The patient had relief from diplopia in the primary position and was not concerned with the doubling still present on dextroversion. Forced ductions still showed marked resistance to forceps rotation into the temporal field.

CASE 2

A 35-year-old white man suffered a skull fracture in an auto accident on December 13, 1956. On recovering consciousness he noted constant double vision. At one month the only sequela was a right sixth nerve paralysis and during succeeding weeks the patient noticed increased "crossing" of the right eye.

Examined on June 10, 1957, vision was 20/20 in each eye. With the paretic right eye fixing he abducted to the midline but with the sound left eye fixing, abduction was short of the midline. Homonymous diplopia was present in all fields. On the troposcope there was fusion at a setting of 50 prism diopters esotropia. A forced duction test on the right eye indicated moderate resistance to forceps rotation into the temporal field.

Electromyography revealed some active innervation of the right lateral rectus though at a much reduced level. During six weeks' observation repeated forced duction tests suggested some further increase in resistance to forceps rotation.

Surgical correction by the Hummelsheim technique was planned as a two-stage procedure. An eight-mm. resection of the right lateral rectus and a four-mm. recession of the right medial rectus were performed on July 18, 1957. At one week there was slight abduction and a small area of fusion at about 25 degrees levoversion. After two months there were 30 degrees of abduction on fixing with paretic right eye. After eight months he could abduct the right eye 45 degrees, and there was single vision on levoversion, primary position, and on dextroversion up to 15 degrees. There was third grade fusion with good amplitude and the patient was completely rehabilitated for work as a machine operator. He was conscious of no disability from the residual diplopia on lateral gaze to the right.

COMMENT

These cases illustrate the importance of contracture in traumatic sixth nerve paralysis. In the neglected case with severe contracture it was apparent that tendon transplants could accomplish little against the shortened medial rectus. In the second case the onset of contracture was taken as the indication for proceeding with surgery. Because electromyography had indicated some nerve function, the horizontal surgery was done as the initial procedure. The results from the resection-recession have been sufficiently satisfactory that the projected tendon transplants have been indefinitely postponed. In retrospect it would seem likely that an even better result might have been obtained in this case if the combined Hummelsheim procedure with tendon transplants had been completed as one operation.

DISCUSSION

PREOPERATIVE EVALUATION

Under ideal circumstances the same ophthalmologist should follow through with a given case of sixth nerve paralysis from the time of injury until the treatment is completed. Unfortunately this is seldom possible. Many of these patients have already made the rounds of several practitioners or have been kept under wraps by a well-intentioned neurosurgeon, and have had a liberal application of the "wait and see" policy. Time must be allowed for possible regeneration of the nerve, but this is not a fixed period. O'Connor stated that if a paralysis was still complete at the end of three months the prospect of spontaneous cure was too slight to warrant further delay, and that he had seen contracture as early as three weeks after injury. Spaeth stated, in 1953, however, that

in head injury cases it was reasonable to wait at least 12 months to allow for spontaneous recovery. Rather than to observe any arbitrary waiting period, it would seem more important that systematic observations be made at regular intervals in order to determine what to do and when to do it.

Careful objective measurements should be made in primary position and on dextroversion and levoversion. The comparative horizontal rotations are important. The perimeter and corneal reflex test is helpful in measuring the degree of rotation but care must be taken that the proper position of the head be maintained with reference to the center of the perimeter. In order that the observations may be significant there must be a notation as to the eye used for fixation and it is preferable that readings be made with the paretic eye fixing and with the sound eye fixing. At best, the objective evaluation in paralytic squint is rather gross and lacks the precision of the prism and cover test which is so valuable in comitant strabismus. Moving pictures afford a rather simple method of recording the progress of paralytic squints.

Diplopia fields are of less help in preoperative study of these more complete sixth nerve paralyses because of the wide separation of the images and the absence of any area of fusion.

O'Connor[17] urged the routine use of a forced duction test at regular intervals. This was recommended as an office procedure under topical anesthesia in order to detect any increased resistance to forceps rotation. The onset of contracture was his prime indication for immediate surgical intervention. Bielschowsky,[18] Reinhardt,[19] Berens, and McLean have all placed emphasis on contracture as an obstacle to the attainment of satisfactory functional results in the correction of traumatic lateral rectus paralysis.

Electromyography has proven a most valuable asset in the study of paralytic lesions of the ocular muscles. Breinin's[20] work on electrophysiology has added greatly to our knowledge of many of the basic problems in motility. To quote Breinin,[21] however, "electromyography is not a tool for office use, nor is it suitable to any and all institutions." He points to the many pitfalls that may be encountered in technique and interpretation. If excellent facilities for recording and interpreting electromyograms happen to be available, they should certainly be used. Breinin[22] states, however, that the presence of some active innervation to a lateral rectus does not insure a functional recovery of that muscle. In his opinion it is not necessary to go to great ends to obtain electromyograms in the evaluation of routine clinical cases of sixth nerve paralysis.

Choice of surgery

As to the choice of surgery, it would seem that no single procedure is applicable to all traumatic sixth nerve paralyses. When no recovery of nerve function is anticipated, as in the case cited by McLean, or when such a long interval has passed that spontaneous recovery seems unlikely, it would seem that the combined Hummelsheim procedure is indicated. Just as a more effective correction of a maximal comitant squint is expected with a full resection and recession at one stage, I believe that in this instance a greater effect may be obtained by performing the combined horizontal surgery and vertical tendon transplant at the same operation.

Girard's recent report of uveitis following the complete detachment of the four rectus muscles would seem to favor the original Hummelsheim technique in which the vertical recti are not completely detached from the globe. This might also be interpreted as a recommendation for the two-stage operation, although I personally have never encountered complicating uveitis in performing either clinical or experimental tendon transplant operations. Surely, if there is even slight function of the paretic muscle with no definite contracture, or if there is indication of some active innervation, the two-stage Hummelsheim procedure would be preferred

as has been suggested by Scobee, Spaeth, and Krewson.

In a neglected case with severe contracture, the recession of this shortened medial rectus muscle is the important step and this should probably be combined with a full resection of the paralyzed lateral rectus. Particularly if binocular vision is recovered in primary position by the resection and recession, and the forceps rotation into the temporal field is still limited, it would seem that further surgery is inadvisable.

Timing of Surgery

As to when to operate, it is agreed that one should proceed when there is no chance for spontaneous recovery of function. As has been suggested, this is not always easy to determine.

Where there is no contracture, the chief consideration may be the patient's annoyance with his appearance and disability due to diplopia. Surgery in this group can be scheduled at convenience. On the other hand, if repeated examinations reveal an increasing deviation or a rising resistance to forceps rotation, surgical interference becomes urgent. To gain a good functional result the operation must anticipate the development of contracture.

Summary

In the surgical treatment of traumatic lateral rectus paralysis, the development of contracture in the opposing medial rectus is a major obstacle to the attainment of good abduction. The onset of this contracture is the prime indication for surgical intervention and this may be determined by frequent measurements of the angle of deviation, careful observation of the rotations, and particularly by repeated forced duction tests.

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References

1. Hummelsheim, E.: Ueber Sehnentransplantation am Auge. Tr. 34, Ophth. Kongress, Heidelberg, 1907, p. 248.
2. ———: Weitere Erfahrungen mit partieller Sehnenuberpflanzung. Arch. f. Augenh., 62-71, 1908-1909.
3. O'Connor, R.: Ocular tendon transplantations. Arch. Ophth., **5**:209, 1931.
4. ———: Tendon transplantation in ocular muscle paralyses. Am. J. Ophth., **18**:813, 1935.
5. Berens, C., and Girard, L. J.: Transplantation of the superior and inferior rectus muscle for paralysis of the lateral rectus. Am. J. Ophth., **33**:1041, 1950.
6. Hill, H. F.: Absence of the medial rectus and its successful treatment by vertical tendon transplants. Tr. Am. Ophth. Soc., **53**:115, 1955.
7. Berens, C.: In discussion of Hill,[6] p. 121.
8. McLean, J. M.: In discussion of Hill,[6] p. 123.
9. Braley, A. E.: In discussion of Hill,[6] p. 124.
10. Girard, L. J., and McNeely, R. A.: Agenesis of the medial rectus muscle. Arch. Ophth., **59**:337, 1958.
11. Scobee, R. G.: The Oculorotary Muscles, Philadelphia, Lea, 1946.
12. Spaeth, E. G.: Surgical aspects of defective abduction. Arch. Ophth., **49**:49, 1953.
13. Krewson, W. E.: Ocular tendon transplantation: indications, variations, and technic. J. Internat. College Surgeons, **27**:731, 1957.
14. Chamberlain, W. P.: Ocular motility in the horizontal plane. Tr. Am. Ophth. Soc., **52**:751, 1954.
15. Hildreth, H. R.: The tendon-transplanting operation. Am. J. Ophth., **36**:1267, 1953.
16. Adler, F. H.: Physiology of the Eye. St. Louis, Mosby, 1950.
17. O'Connor, R.: Contracture in ocular muscle paralysis. Am. J. Ophth., **26**:69, 1943.
18. Bielschowsky, A.: Lectures on motor anomalies; paralysis of individual eye muscles, abducens—nerve paralysis. Am. J. Ophth., **22**:357, 1939.
19. Reinhardt, P. H.: Correction of external-rectus paralysis with contracture of the opposing internus. Am. J. Ophth., **27**:636, 1944.
20. Breinin, G. M.: Electromyography—a tool in ocular and neurologic diagnosis: II. Muscle palsies. Arch. Ophth., **57**:165, 1957.
21. ———: New aspects of ophthalmoneurologic diagnosis. Arch. Ophth., **58**:375, 1957.
22. ———: Personal communication.

SEGMENTAL RETINAL PERIARTERITIS*

A REPORT OF THREE CASES

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Segmental retinal periarteritis is a rare condition of the eyes in which discrete plaquelike exudates are found applied to the retinal arteries. Yellowish white, these lesions encircle the arteries repeatedly, much like beads spaced on a string. This condition is usually associated with a patch of acute exudative choroiditis and tends to disappear as the latter heals. Although several authors[1-3] have associated this condition with tuberculosis, the relationship is by no means constant.

We have added the term "segmental" to "retinal periarteritis" to differentiate it from those conditions in which exudate can be seen coursing parallel to the retinal arteries and their surrounding sheaths, frequently seen in retinal diseases producing fluid exudates, such as retinitis and retinal edema. The segmental type, on the contrary, is rarely seen.

Within the past two years we have seen three cases of segmental retinal periarteritis. Because of its apparent rarity and distinctive appearance, we felt that these cases might prove of interest to others.

A few cases have been reported in the literature. Elwyn[1] remarks on its extreme rarity, quotes only three authors who had observed this condition, and cites no case of his own. Duke-Elder,[2] quoting the same cases mentioned by Elwyn, thought it to be a primary tuberculous condition.

The earliest presentation of this condition that we could find was by Kyrieleis[3] in 1933. He described a man who, following an injury to the right side of his face, developed an acute choroiditis in the right eye. When the media had cleared sufficiently to permit a fundus examination, in addition to the localized acute choroiditis, a series of white ringlike exudates were seen extending the width of the arteries. The lesions were particularly marked where the vessels divided. The veins showed no change. By three months, as the choroiditis healed, they had practically faded away. Because of a positive tuberculin reaction, Kyrieleis thought these lesions to be a local allergic reaction to the presence of tubercle bacilli or their toxins.

In 1939, Muncaster and Allen[4] presented the case of a 31-year-old school teacher who, following a negative tuberculin test, was given a second one which was strongly positive. She developed a stormy uveitis. When the media had cleared, there were seen "small round spots of gray exudates resembling tubercles around the larger arteries near the disc in each fundus." Over the next two months these exudates were gradually absorbed as the eyes returned to normal.

In 1952, Thompson[5] described this condition in a 20-year-old Royal Canadian Air Force officer. This patient, first seen in March, 1944, complained of seeing black spots in front of his eyes for the preceding two months. Examination showed each eye to have an old iridocyclitis with deposits on the lens, posterior synechias, and vitreous opacities. Ophthalmoscopy revealed multiple cufflike white plaques on the retinal arteries, more marked in the left eye. Exhaustive tests for tuberculosis were negative. The only positive finding was an X-ray film which showed both antra to be opaque. During the following 14 months, except for three small ones near the optic disc, the exudates on the retinal arteries disappeared.

CASE PRESENTATIONS

CASE 1

A 25-year-old single identical twin Negro clerical worker was first seen September 13, 1955, complaining of a film over his left eye of one week's duration. He had no history of any previous visual

* From the Jewish Hospital of Brooklyn and the Brooklyn Eye and Ear Hospital.

difficulty, and his health had always been good. General physical examination, except for his eyes, was completely negative.

Eye examination showed his corrected vision to be: O.D., 20/25; O.S., 20/200. The right eye was negative except for several patches of nevoid pigmentation in the temporal area of the retina. The left eye showed a moderate ciliary injection. The aqueous was very hazy and plastic exudates were seen on the cornea and lens. After the pupil was dilated, the iris was seen to be edged with small gray translucent nodules. The vitreous was so dense that the fundus was seen with difficulty. The disc outline could not be made out, the retina seemed very edematous, and there were several hemorrhagic areas with white centers at the posterior pole.

The patient was treated with local atropine, steroids, and antibiotics, and was given 20 mg. Meticorten a day by mouth.

One week later the Koeppe nodules in the left eye had almost disappeared, and the media had cleared sufficiently so that a large exudative lesion of the choroid could be seen at the 4-o'clock position about three disc diameters from the disc margin. It appeared raised and dense, with small blood vessels coursing along the margin. The retina remained edematous, and the blood vessels were very irregular and narrow.

One month later the ciliary injection was gone and the aqueous had cleared. The exudative area had flattened and was slate gray. Applied to the superior and inferior retinal arteries were yellowish plaques, more numerous near the disc. One ensheathed the first division of the inferior retinal artery like a pair of pants (fig. 1).

Three months after onset medication was discontinued. Vision in the left eye had increased to 20/30, and the eye was quiet. The area of choroiditis had a large white center with a gray border surrounded and invaded by fine blood vessels. The plaques had faded but were still visible.

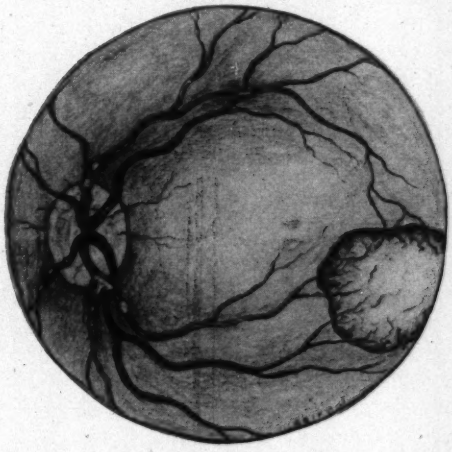

Fig. 1 (Griffin and Bodian). *Case 1.* A plaque ensheathing the first division of the inferior retinal artery like a pair of pants. Segmental retinal periarteritis associated with acute chorioretinitis.

An extensive work-up was negative. The Mantoux test was negative in dilutions of 1:1,000. Chest X-ray films were negative. Within normal limits were hemoglobin, hemocrit, stained blood smear, and B.M.R. Mazzini and urine were negative. Serum agglutination tests for typhoid O.H., Para A and B, Brucella, proteus X19 were negative. Stool was negative for ova and parasites. Kveim test for sarcoidosis was negative.

Final diagnosis: Acute choroiditis with segmental retinal periarteritis. Cause not determined.

Case 2

A 37-year-old married Negress, a factory worker, was first seen at the Brooklyn Eye and Ear Hospital on February 27, 1954. At that time she had a patch of acute chorioretinitis in the right eye. A week later she was seen to have all the retinal arteries of the right eye blighted with discrete yellowish exudates. These were most prominent near the disc. There was a large patch of choroiditis in the left lower temporal quadrant, and the vitreous was hazy. Urine and Kahn were negative. Mantoux 1:10,000 showed very slight reaction in 24 hours. She was treated with 25 mg. cortisone by mouth three times daily. She was last seen at the Brooklyn Eye and Ear Hospital six weeks after admission. The vitreous was still slightly hazy, and the plaques were still present along the arterioles. The area of choroiditis showed some shrinkage.

The patient was next seen when she came into the office on July 9, 1955, complaining of pain in her right eye and a foreign body feeling. She reported no trouble with her eyes since her last visit to Brooklyn Eye and Ear Hospital. She had always been healthy and gave no history of any major illnesses. Except for her eyes, general physical examination was negative.

Examination of the eyes showed a vision of: O.D., 20/40; O.S., 20/15. The right eye had an episcleral and a ciliary injection. The pupil was small and reacted poorly. The aqueous showed many cells and the vitreous was hazy with a veil formation. The fundus showed an area of old chorioretinitis in the lower nasal area. Adjacent to this, a fresh exudative lesion extended linear infiltrations out into the vitreous. Periarterial exudates were not noted at this time.

The patient was treated with local atropine, cortisone, and antibiotics. She was given 20 mg. Meticorten per day by mouth.

One week later the media had cleared enough for better observation of the fundus. Many blocklike areas of yellowish exudates were seen lying on top and surrounding the arteries, even out to the second and third divisions. They were at fairly regular intervals and were of similar size and shape (fig. 2).

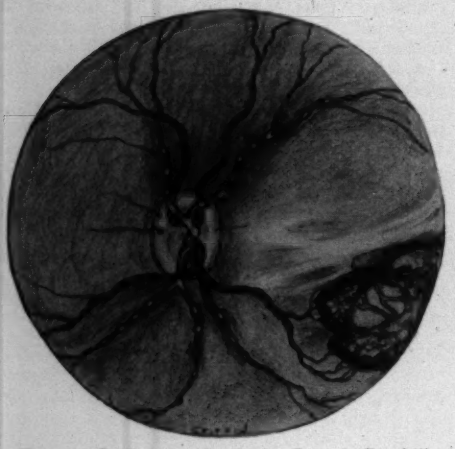

Fig. 2 (Griffin and Bodian). *Case 2.* Blocklike areas of exudates lie on top of and surround the arteries. Note acute chorioretinitis.

One month later the aqueous had completely cleared of cells. The vitreous veil remained, and the periarterial exudates persisted but became more refractile in appearance. Some were more or less absorbed so that they resembled aphids on a stem, some seen directly and some seen sideways. Some lesions had coalesced. The choroidal exudation had flattened, and the infiltrations into the vitreous had receded. The pigmentation of the choroidal lesion had increased and the center had thinned so that some of the choroidal vessels could be seen.

During the following three months the patient had two slight flare-ups, and again six months later. Since then the eye has remained quiet without medication. The vitreous veil receded somewhat. The choroidal lesion showed still more pigmentation. The periarterial exudates were still present, but fading, and were brightly refractile in spots.

Chest X-ray films were negative. Mantoux 1:10,000 was slightly positive after 24 hours. Mazzini and urine tests were negative, as were tests for brucellosis, typhoid, paratyphoid, and for intestinal ova and parasites.

Final diagnosis: acute chorioretinitis with segmental retinal periarteritis, cause not determined.

Case 3

A 26-year-old unmarried Negress, a cosmetic worker, was seen on June 27, 1955, complaining of pain, redness, photophobia, and diminished vision in her left eye for six days. The onset had been sudden and without apparent cause. Aside from her eye condition she was completely well. A general physical examination revealed no pathologic condition.

Eye examination showed the vision in the right eye to be 20/15. No pathologic process was present. The vision in the left eye was 20/30, not correctible. There were moderately severe ciliary injection, numerous gray keratic precipitates, the largest of which measured about one mm., and a hazy aqueous containing floating clumps of cells and exhibiting a marked flare. The iris stromal structure was slightly indistinct. The pupil, three mm. in diameter, reacted promptly to light. The vitreous was slightly murky throughout with a few threadlike opacities floating freely. All landmarks in the fundus were hazy. The inferior nasal and temporal arteries were studded with yellowish plaques along their course from disc margin to periphery. The plaques were most numerous over the temporal vessels and most prominent near the disc. The superior vessels showed no segmental exudates. About five disc diameters from the nerve-head near the inferior temporal vessels was a large patch of heaped-up bright white exudate surrounded by edema of the retina. This measured three disc diameters in size and had the typical appearance of an acute chorioretinitis (fig. 3).

Laboratory work-up showed blood Kahn and urine to be negative.

On hot compresses, atropine, and hydrocortisone drops, the iritic reaction subsided considerably. Hydrocortisone, 20 mg. by mouth, was started because the choroiditis showed no response. By September, 1955, her vision was 20/15 in each eye. The keratic precipitates were shrunken and pigmented. The choroiditis was no longer acutely active. The area had flattened, and was being surrounded by increasingly pigmented margins. The inferior nasal arteries had lost their segmental exudates, and those on the inferior temporal vessels had faded greatly. Both inferior arterioles were now overlaid by a streaked tunic of edema (fig. 4).

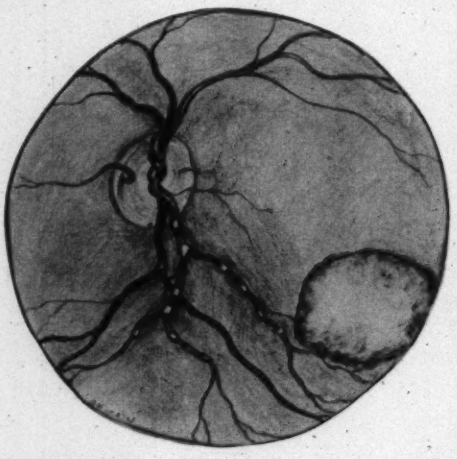

Fig. 3 (Griffin and Bodian). *Case 3.* A large patch of heaped-up bright white exudate was surrounded by edema of the retina. Segmental periarteritis confined to lower retinal vessels. Associated acute chorioretinitis.

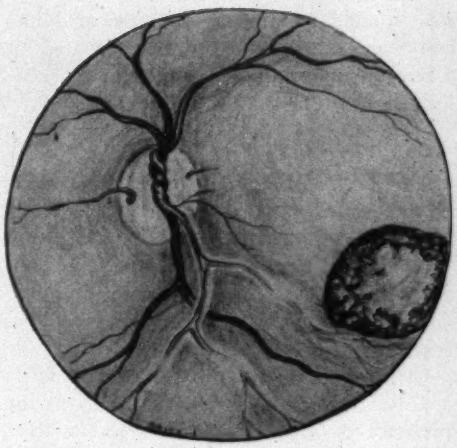

Fig. 4 (Griffin and Bodian). *Case 3,* three months later. Both inferior arterioles were overlaid by a streaked tunic of edema.

On October 26, 1955, the streaked sheathing of the vessels was practically gone; however, a suggestion of the plaques still remained over the inferior temporal arteriole. The choroiditis seemed completely healed (fig. 5).

Final diagnosis: acute chorioretinitis with segmental retinal periarteritis, cause not determined.

Discussion

Judging from the literature, segmental retinal periarteritis is extremely rare. Only three cases have been reported. However, we have seen three cases within a two-year period. Why the discrepancy? There are two possible answers. Either we were simply fortunate or the condition is not frequently recognized. Perhaps cloudy media mask the phenomenon in the early stages of choroiditis. In the healing stages the arteries may clear before the media so that the segmental exudates cannot be observed. This would account for failure of observers to record the finding more often.

The incidence of this condition is interesting. All three cases were found in young adult Negroes (one male, two females) who were in apparent good health aside from the eye lesions. In all cases the segmental periarteritis was monocular and was associated with a patch of acute exudative choroiditis which responded to corticosteroids. As the choroiditis cleared, the segmental periarteritis diminished.

The older literature describes segmental periarteritis as a tuberculous infection of the eye. However, this study would cast doubt on such a conclusion. To begin with, none of the patients whom we presented showed clinical evidence of tuberculosis elsewhere in the body. Even the tuberculin tests were inconclusive or negative. In all cases the choroiditis responded to systemic doses of corticosteroids. Had the lesions been frankly tuberculous, we should not expect the lesions to clear with these agents. And yet they did. We must therefore conclude that these cases of acute choroiditis with segmental periarteritis were not of tuberculous origin.

Although no other specific cause can be established for the periarteritis, one fact was clear. In our cases it was always associated with a patch of acute exudative choroiditis. The arterial exudates were largest and most numerous when the choroiditis was at its peak and faded as the choroiditis healed. Quite possibly the plaques on the vessels represented migration of exudate from the active choroiditis to the periarterial sheaths.

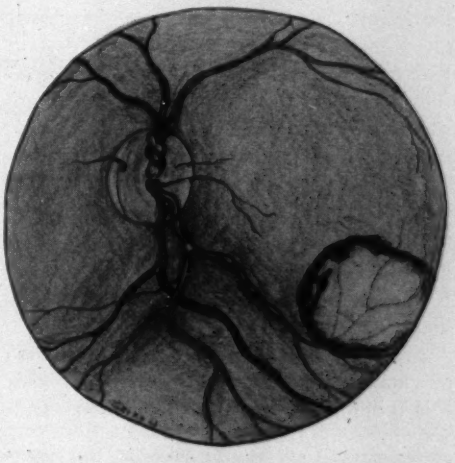

Fig. 5 (Griffin and Bodian). *Case 3,* four months later. A suggestion of the plaques still remained over the inferior temporal arteriole.

The reason for the segmental distribution of the periarteritis can only be conjectured since no pathologic reports are available. Possibly it is due to a peculiar anatomic arrangement in some individuals of the perivascular sheaths into compartments. If some of these compartments were more permeable than others, one could understand how exudate could enter some segments of the sheath and not others. The rarity of segmental periarteritis may be due to the infrequency of such an anatomic anomaly being coupled with an exudative chorioretinitis.

Another explanation of its segmental arrangement may be that the lesions are inflammatory allergic reactions. Such reactions are known to have a spotty distribution on occasion, for example, hives. Further evidence for the allergic nature of the segmental periarteritis lies in the fact that it cleared in response to corticosteroid therapy. Allergy is also brought to mind by the case of Muncaster and Allen[4] which was precipitated by a second subcutaneous injection of tuberculin given for diagnostic purposes.

Summary and Conclusions

Three cases of segmental retina periarteritis are described. Reasons for adding "segmental" to "retinal periarteritis" are given. These lesions were unilateral, associated with a patch of acute exudative chorioretinitis, and occurred in young Negro adults. All responded to systemic corticosteroids. The most likely cause for the periarteritis is an allergic reaction of unknown origin in the presence of acute exudative retinitis. Reasons for the segmental distribution of exudate are suggested.

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References

1. Elwyn, H.: Diseases of the Retina. New York, Blakiston, 1953, ed. 2, p. 498.
2. Duke-Elder, W. S.: Textbook of Ophthalmology. St. Louis, Mosby, 1941, v. 3, p. 2672.
3. Kyrieleis, W.: Ueber atypische Gefässtuberkulose der Netzhaut. Arch. f. Augenh., **107**:182, 1933.
4. Muncaster, S. B., and Allen, H. E.: Bilateral uveitis with retinal periarteritis as a focal reaction to the tuberculin test. Arch. Ophth., **21**:509, 1939.
5. Thompson, A. M. W.: Periarteritis retinae. Brit. J. Ophth., **36**:268, 1952.

CATARACT FOLLOWING GLAUCOMA SURGERY*

Inferior approach—scleral incision

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The proper placement of an incision to extract a cataractous lens following a fistulizing operation for glaucoma should be performed with a clear understanding of its limitations.

In 1954, I[1] reported on a preferred method of cataract extraction in which an inferior limbal approach was used. The lens was extracted in capsule by retracting the iris and using a sliding Verhoeff technique. The present study comprises a review of 22 cases in which a similar procedure was followed with slight modifications.

Altered physiologic considerations

An eye which has undergone a filtering procedure for the control of increased tension can be considered to be functioning under abnormal conditions. The manner in which the tension is controlled is still a ques-

* From the Holy Family Hospital, St. Peter's Hosiptal, and the Brooklyn Eye and Ear Hospital. Presented before the Pan-American Association of Ophthalmology, February, 1958.

tion of considerable controversy. The mechanical filtration through the newly produced scleral opening or the readjustment of the neurovascular status as a result of a reflex mechanism, due to the surgical trauma of the uveal tract, has their advocates. The altered anatomic and physiologic changes that have occurred modify the prognosis and often complicate the problem of the cataract extraction.

The filtering cystic scar may be extensive and encroach into corneal tissue to the degree that it complicates lens extraction in that locality. In some cases, hypotony may result. Anterior adhesions of iris or vitreous to the posterior surface of the cornea superiorly can produce varying degrees of dystrophic changes or corneal scarring.

If the anterior chamber remains shallow postoperatively, a subsequent Graefe knife section may produce complications. Posterior synechias are frequently encountered and require careful separation with a thin spatula to facilitate extraction of the lens intracapsularly. An updrawn pupil occasionally results from an iridencleisis and may require a sphincterotomy. The anterior lens capsule is often found to be fragile, especially in intumescent cataracts. A motor-driven or hand erisophake should then be used in preference to the lens forceps.

In the occasional case where a lens has been subluxated from previous trauma due to glaucoma surgery, it may be judicious to employ a Weber loop for its removal. Although at times the lens may be found to be adherent to the vitreous, intracapsular extraction is usually accomplished with surprising ease. This can be explained on the basis of degenerated weakened zonular fibers.

In spite of fluid vitreous which is usually present as a result of uveal trauma, vitreous loss is indeed rare in this type of cataract extraction. This can be attributed to the condensation of the anterior face of the hyaloid membrane which is frequently encountered. The visual prognosis should always be guarded for it is dependent not only on the surgical technical difficulties experienced, but to the degree of optic nerve damage that may have occurred from the pre-existing glaucomatous condition or macular degeneration.

Surgical considerations

The incision for the removal of cataract could be placed superiorly, either through or below the filtration area; temporally or inferiorly. A section through the filtration area is seldom used unless the filtering bleb produces a state of hypotony. In a study of 91 eyes with fistulizing operations, DeVoe[2] obtained approximately 15 percent of grossly hypotonic eyes. Meyer[3] prefers this method of extraction although he is aware of the great danger of excessive postoperative fibrosis which can result in closure of the filtration area with recurrence of the glaucoma. The advantage of this method is the familiarity in making the corneal section in the usual standard fashion.

Recent papers have appeared by Scheie[4] and by McPherson and Fisher[5] who advocate a corneal incision just below the filtering bleb. Scheie reported on 22 eyes covering a period from 1951 to 1955 in which the incision was made perpendicular in clear cornea one mm. below the bleb. Vitreous was lost in two eyes; one eye resulted in an updrawn pupil and one eye developed a postoperative iridocyclitis. Satisfactory results were obtained in the remaining 18 cases.

In the more recent paper McPherson and Fisher in a series of 24 eyes covering the years 1945 to 1956 used a similar surgical approach. They reported rupturing the anterior capsule in 10 of their cases and vitreous was lost in three. They stated that in three of the 24 cases poor visual acuity was obtained which they attributed to faulty surgical technique.

A superior placed incision in clear cornea produces an overhanging corneal shelf that can make the extraction of the cataract rather difficult. Furthermore, the adhesions between iris and vitreous to the posterior surface of the superior portion of the cornea can fur-

TABLE 1

DATA ON CATARACT EXTRACTION FOLLOWING FILTERING GLAUCOMA OPERATIONS
(22 CASES—USE OF INFERIOR LIMBAL APPROACH)

Eye	Age Sex	Previous Glaucoma Surgery	Intracapsular Lens Extraction	Complications Following Surgery	Ocular Tension	Remarks Corrected Vision
1	64—F	Mar. 1945 Trephination	Oct. 1952 Verhoeff sliding method	None	Normal	20/70 Previous glaucomatous cupping
2	61—F	June 1952 Iridencleisis	Mar. 1953 Tumbling method with forceps	None	Normal	20/20
3	72—F	Jan. 1940 Trephination	Mar. 1953 Tumbling method with forceps	Temporary striate keratitis	Normal	20/25
4	38—M	Feb. 1953 Iridencleisis	Mar. 1953 Tumbling method with Bell erisophake	Secondary rise in intraocular pressure	Normal	20/25 Cyclodialysis performed April 1953
5	69—F	Mar. 1953 Iridencleisis	May 1953 Verhoeff sliding method	Striate keratitis cleared after 3 wk	Normal	20/40
6	67—F	Feb. 1953 Iridencleisis	May 1953 Extracapsular	Slow absorption of lens cortex	Normal	10/200 Capsulotomy performed 6 mo. later
7	69—F	Mar. 1953 Iridencleisis	Oct. 1953 Tumbling method with Bell erisophake	Striate keratitis cleared after 4 wk	Normal	20/30
8	77—F	June 1951 Iridencleisis	Sept. 1953 Tumbling method with forceps	Slight vitreous loss	Normal	8/200 Previous optic atrophy existed
9	60—M	Aug. 1953 Iridencleisis	Oct. 1953 Verhoeff sliding method	Slight striate keratitis cleared after 5 da.	Normal	20/25
10	66—F	Feb. 1952 Iridencleisis	Jan. 1954 Verhoeff sliding method	None	Normal	20/200 Previous optic atrophy existed
11	72—F	May 1953 Iridencleisis	Jan. 1954 Verhoeff sliding method	None	Normal	20/70 Previous optic atrophy existed
12	69—F	June 1950 Iridencleisis	Jan. 1954 Verhoeff sliding method	None	Normal	20/30
13	53—F	Jan. 1954 Iridencleisis	Apr. 1954 Extracapsular extraction. Weber loop used	Severe postoperative reaction	Elevated	3 Cyclodiathermy procedures performed absolute glaucoma eventually developed June 1957
14	65—F	May 1954 Iridencleisis	Sept. 1954 Verhoeff sliding method	None	Normal	20/200
15	54—M	Mar. 1954 Iridencleisis	Jan. 1955 Verhoeff sliding method	None	Normal	20/40—
16	64—F	Dec. 1952 Iridencleisis	Feb. 1955 Verhoeff sliding method	Slight striate keratitis cleared after 1 mo	Normal	20/70 Some recurrence posterior synechias

TABLE 1 *(Continued)*

Eye	Age Sex	Previous Glaucoma Surgery	Intracapsular Lens Extraction	Complications Following Surgery	Ocular Tension	Remarks Corrected Vision
17	69—F	Dec. 1955 Iridencleisis	Apr. 1956 Barraquer erisophake used	Pronounced striate keratitis	Elevated	5/200 Bullous keratopathy developed
18	63—M	Feb. 1957 Iridencleisis	June 1957 Verhoeff sliding method	None	Normal	20/20+
19	63—F	June 1957 Iridencleisis	Sept. 1957 Extracapsular extraction	Little cortex remained. Was well absorbed later	Normal	20/70 Previous macular degeneration
20	69—F	Mar. 1956 Iridencleisis	Oct. 1957 Verhoeff sliding method	None	Normal	20/30—
21	60—F	Aug. 1953 Iridencleisis	Nov. 1957 Verhoeff sliding method	None	Normal	20/40— Partial optic atrophy
22	63—F	Oct. 1956 Iridencleisis	Apr. 1958 Verhoeff sliding method	None	Normal	20/20

ther add to the surgical difficulties in this area.

The temporal inferior incision used by some surgeons has not been popular for several reasons. Meyer,[6] using this approach, experienced bullous keratopathy of an intractable nature in four cases. He may have made his incision, however, in clear cornea which could have endangered the corneal metabolism. The corneal diameter is elongated in the transverse direction and lens extraction thus becomes more awkward. Furthermore, to facilitate removal of the lens, an iridectomy is usually required which adds to increased bleeding and further mutilation.

The inferior aproach has been preferred by Kronfeld,[7] Castroviejo, Sourdille,[8] and François.[9] It is the method of choice for me. Due to its location, the procedure may at first appear to be somewhat awkward; however, the average surgeon has little difficulty in making the proper adjustment.

Surgical technique

1. To prevent nausea and postoperative vomiting, Vesprin 20 mg. (1.0 cc.) is administered intramuscularly one hour before surgery; 75 to 100 mg. Demerol is given intravenously one-half hour before surgery.

2. A Guyton-Park speculum is used to avoid pressure on the globe.

3. A small limbal-based conjunctival flap is made from the 3- to 9-o'clock meridian inferiorly.

4. With use of Bard-Parker knife and No. 111 blade, a small scleral incision, 1.5 mm. from the limbus, is made at the 4- and 8-o'clock meridians. Bleeding is controlled with the use of a pointed heated cautery or by the instillation of 10-percent Neosynephrine.

5. Two McLean type sutures are put in place using braided 6-0 black silk sutures.

6. A narrow keratome incision is introduced at the 6-o'clock meridian in sclera 1.5 mm. from the limbal area. The corneal wound is enlarged with scissors.

7. Posterior synechias are carefully separated with the use of a thin spatula by gently lifting the cornea.

8. Very slight point pressure is applied at the superior limbal area adjacent to the area of filtration.

9. The iris is retracted carefully with a small muscle hook or special retractor.[10] In cases of updrawn pupil, sphincterectomy is

performed with straight scissors.

10. The lens is extracted intracapsularly, preferably with the use of a lens forceps by the sliding Verhoeff technique.

11. At the completion of the operation, air is injected in the anterior chamber by using a bent, blunt, 30-gauge needle.

12. If tight closure of the corneal wound has not been accomplished, additional corneoscleral sutures are put in place.

In intumescent cataracts, the use of an erisophake may be required. The tumbling technique is not advisable because of the undue trauma that may result to the endothelial surface of the cornea which may precipitate bullous keratopathy. Guyton[11] stressed the importance of making a scleral incision in cataract extraction when corneal dystrophic changes are suspected. McLean[12] also emphasizes this type of surgical approach and, in suspected cases of Fuchs' dystrophy, he employed a short inferiorly placed shelving scleral incision and removed the lens in capsule by the sliding method.

By using a scleral incision, I am also of the opinion that less disturbance in corneal metabolism results.

Survey of cases

A report follows on 22 eyes in which cataract extraction was performed by means of an inferior approach and which covered a period from 1952 to 1958. In all cases, the ocular tension was controlled by means of a fistulizing operation. The first 12 eyes were operated by means of a limbal incision and in the remaining 10, scleral incision was used and the technique followed was that already outlined (table 1).

In this series, there were 18 females and four males. Iridencleisis had been previously performed in 20 eyes and corneoscleral trephination in two eyes. Intracapsular extraction was successfully performed in 19 eyes. One complication resulted which eventually led to an intractable bullous keratopathy with marked reduction of vision. This was evidently the result of endothelial damage from excessive intracameral instrumentation. The postoperative complications are outlined in Table 2.

TABLE 2

Postoperative complications (22 eyes)

Extracapsular	3
Vitreous loss	1
Striate keratitis	5
Visual loss	2
Increased tension	3

In the three eyes that extracapsular extraction inadvertently occurred, two had retained lens cortex at the completion of the operation, but excellent absorption followed with a minimal amount of reaction. One eye required a capsulotomy which when performed six months later resulted in improvement of vision. In the third eye, a considerable amount of lens cortex remained, and a severe postoperative reaction followed, which simulated a phacoanaphylactic reaction. This eye eventually ended in absolute glaucoma in spite of three cyclodiathermy procedures which were performed to control tension. Loss of fluid vitreous occurred in one eye which produced no undue complications. In five eyes, striate keratitis was seen following surgery but in four, complete clearing resulted within a period of one month. In three eyes that showed increased tension, one developed absolute glaucoma and another bullous keratopathy. In the third case, the tension was later controlled by cyclodialysis.

In conclusion, the operative results were satisfactory. Improvement in vision was obtained in 20 eyes and the cosmetic appearance in these eyes was gratifying. Some of these cases are illustrated in this article (figs. 1 through 7).

Summary and Conclusions

1. This paper is intended to be a supplementary report of my preferred method of cataract extraction following successful filtering glaucoma operations. A review of 22 eyes is presented covering a period from 1952 to 1958. An inferior limbal approach was used

Fig. 1A (Rizzuti). Fistulizing procedure with controlled tension and cataract formation.

Fig. 1B (Rizzuti). Cataract extraction; no dystrophic changes of cornea. Filtering bleb functioning; tension normalized without miotics.

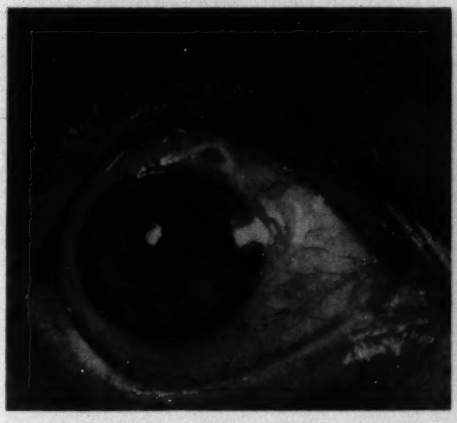

Fig. 2A (Rizzuti). Fistulizing procedure with controlled tension and cataract formation.

Fig. 2B (Rizzuti). Cataract extraction; no dystrophic changes of cornea. Filtering bleb functioning; tension normalized without miotics.

Fig. 3A (Rizzuti). Fistulizing procedure with controlled tension and cataract formation.

Fig. 3B (Rizzuti). Cataract extraction; no dystrophic changes of cornea. Filtering bleb functioning; tension normalized without miotics.

Fig. 4A (Rizzuti). Fistulizing procedure with controlled tension and cataract formation.

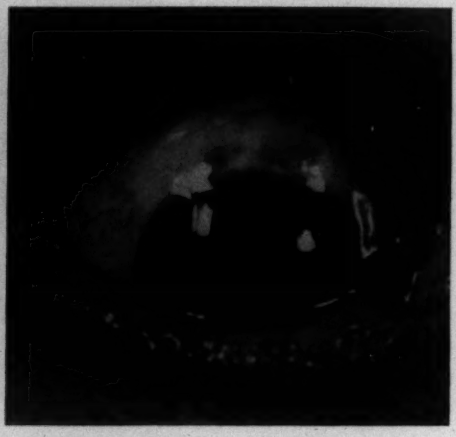

Fig. 4B (Rizzuti). Cataract extraction; no dystrophic changes of cornea. Filtering bleb functioning; tension normalized without miotics.

Fig. 5A (Rizzuti). Fistulizing procedure with controlled tension and cataract formation.

Fig. 5B (Rizzuti). Cataract extraction; no dystrophic changes of cornea. Filtering bleb functioning; tension normalized without miotics.

in all cases. Since 1954, 10 cases were operated by using a scleral incision at the 6-o'clock meridian, 1.5-mm. from the corneal limbus. The iris was retracted after severing posterior synechias. The Verhoeff sliding technique, removing the lens in capsule, was the method of choice.

2. The advantages and disadvantages of other types of incisions employed were discussed.

3. It has been stressed that loss of vitreous and retained lens cortex could lead to deleterious effects resulting in absolute glaucoma.

4. A scleral type of incision is believed to minimize the dangerous complications of partial or total bullous keratopathy.

5. With greater longevity, filtering glaucoma procedures complicated by cataract will be on the increase. Early peripheral iridectomy or iridotomy as a prophylaxis, or to combat the initial attack of glaucoma, may

Fig. 6A (Rizzuti). Fistulizing procedure with controlled tension and cataract formation.

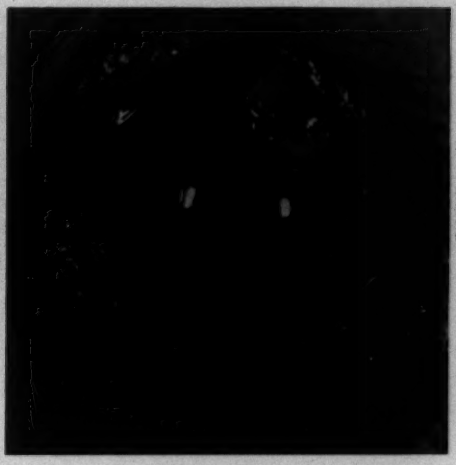

Fig. 6B (Rizzuti). Cataract extraction; no dystrophic changes of cornea. Filtering bleb functioning; tension normalized without miotics.

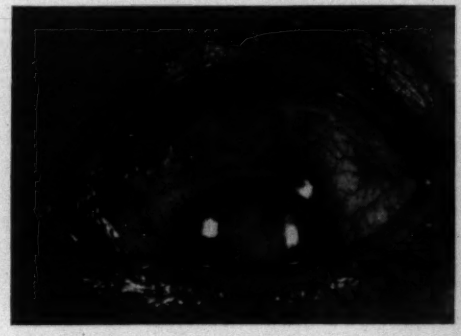

Fig. 7A (Rizzuti). Fistulizing procedure with controlled tension and cataract formation.

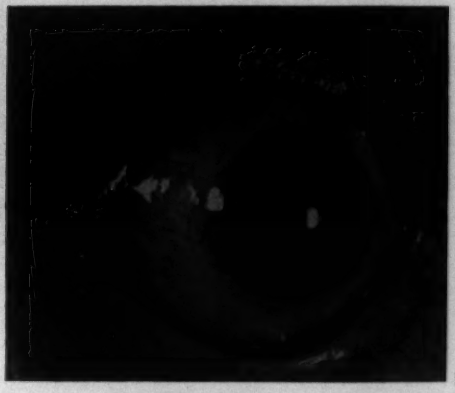

Fig. 7B (Rizzuti). Cataract extraction; no dystrophic changes of cornea. Filtering bleb functioning; tension normalized without miotics.

lessen some of the complications in cases that later require lens extraction.

6. For a more accurate study in tabulating statistics in this special type of surgery, the cataract extraction should be performed by the same operator. In the final analysis the method employed should be the one that is particularly suited to the surgeon.

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References

1. Rizzuti, A. B.: Cataract extraction following fistulizing glaucoma operations: Method of choice by inferior limbal approach. Acta XVII Conc. Ophth., 1954, **3**:1831, 1955.
2. DeVoe, A. G.: A gonioscopic study of fistulizing operations. Arch. Ophth., **46**:504-509 (Nov.) 1951.
3. Meyer, S. J.: Personal communication.
4. Scheie, H. G.: A method of cataract extraction following filtering operations for glaucoma. Arch. Ophth., **55**:819-829 (June) 1956.

5. McPherson, S. D., and Fisher, G. W.: Results of cataract extraction following filtering operations for glaucoma. Southern M.J., **51**:99-102 (Jan.) 1958.
6. Meyer, S. J. and Sternberg, P.: Corneal dystrophy following secondary intraocular operations. Arch. Ophth., **46**:527-530 (Nov.) 1951.
7. Kronfeld, P. C.: Personal communication.
8. Sourdille, G. P., and Hervouet, F.: Inferior corneal incision in the operation for cataract after fistulizing intervention. Bull. Soc. ophth. Paris, Mar., 1949, pp. 543-545.
9. François, J.: Glaucoma and cataract. Ann. d'ocul., **180**:457 (Aug.) 1947.
10. Rizzuti, A. B.: An iris retractor and expressor for round-pupil intracapsular cataract extraction. Am. J. Ophth., **37**:941 (June) 1954.
11. Guyton, J. S.: Prevention and management of complications of cataract extraction. Am. J. Ophth., **31**:1085-1096 (Sept.) 1948.
12. McLean, J. M.: Extraction in complicated cases. Tr. Am. Acad. Ophth., **58**:371-377, 1954.

THE FUSION FREQUENCY OF FLICKER AS A CRITERION OF CENTRAL NERVOUS SYSTEM FATIGUE*

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Most of the physiologic fatigue research has been concerned with physical work. The experimental basis is well established; the load can be determined in terms of meter-kilograms or equivalent units, and there are large changes of metabolic, respiratory, and cardiovascular functions which can be easily and accurately measured. The cardiovascular or respiratory reserve capacity is well defined. Yet, in the present trends of development in industry and military occupations, the emphasis has entirely shifted to fatigue of the central nervous system, with the progressive elimination of muscular effort. Measurement of central nervous fatigue is infinitely more complicated. Work performance is an unreliable index of fatigue, since it depends also on motivation. The common assumption that fatigue is proportional to the length or intensity of work or the subjective effort is not necessarily true. Reading, for instance, which has been used in "fatigue" studies, may actually be relaxing, even when continued for several hours. The metabolic or physicochemical changes in the brain are not accessible to measurement, and they are too small to affect significantly the concentration of metabolites in the blood, although the local metabolic rate in the central nervous system is high. In short, there is no reliable, objective, and independent criterion available for evaluation of any test for fatigue of the central nervous system. There is, however, a universal feature of fatigue, observed in all functions accessible to precise measurement: the decrease of excitability.

It is for this reason that I tried to apply the fusion frequency of flicker for fatigue studies. The fusion frequency of flicker is related to the time parameters of excitability, that is, to the latent period, the activation time, and the refractory period. Of these phases, the refractory period is probably the most important.[1] Excitability is affected by a variety of conditions, such as hypoxia, acidosis, disturbance of the metabolism, and electrolyte equilibrium. A decrease of the fusion frequency of flicker, therefore, is not specific.

The starting point for use of the fusion frequency of flicker as a fatigue test was my observation[2] of a highly significant drop of 5.0 flashes/sec. in 17 subjects during eight hours of sedentary work (laboratory and clerical work).

Instead of the usual decrease of the fusion

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frequency of flicker during the working day, the fusion frequency of flicker increased after amphetamine[3] and pervitin[4]; taken late in the morning. The effect of amphetamine (mean of four subjects: + 9.5 c.p.s.) and pervitin (mean of 16 subjects: + 5.5 c.p.s.), calculated as difference between the days with and without the drugs, was statistically highly significant, and paralleled the removal of subjective fatigue. A similar stimulating effect was later observed by Schmidtke,[5] by Wachholder and Schneider[6] for caffeine, and by Larson, et al.,[7] after smoking a cigarette.

In contrast, alcohol depressed the fusion frequency of flicker, parallel to the concentration in blood, but without correlation to the subjective effects. It was concluded that the fusion frequency of flicker is an objective test for the state of the excitabiity of the central nervous system rather than reflecting the subjective sensation of fatigue. Schmidtke[5] found a depression of the fusion frequency of flicker starting 20 to 30 minutes after intake of 50 to 150 mg. Phanodorm, parallel to the dose but not to the subjective sensation of tiredness. These results support our conception that the fusion frequency of flicker is related to excitability.

In our study on laboratory and clerical workers, we found no significant difference in the drop of the fusion frequency of flicker between occupations with high (microscopy) and low visual effort. We assumed that the decrease of the fusion frequency of flicker is related to the state of the central nervous system rather than to the receptors in the retina. This was more specifically shown by investigation of strenuous inspection work, involving recognition of moving small letters with a critically short exposure time, performed under a wide range of illumination from two to 300 foot-candles for a duration of two hours.[9]

The drop of performance, which is in experimental subjects with high level of motivation the most meaningful fatigue index, was least pronounced at 100 foot-candles in six subjects (mean decrease, 5.7 percent) and most pronounced at the grossly inadequate level of two foot-candles (mean decrease, 20 percent). The difference in the drop between two foot-candles and 100 foot-candles, which parallels the subjective difficulty of the visual task, was statistically highly significant. There was a slight (about one flash) but statistically highly significant decrease of the fusion frequency of flicker, which was, however, uniform for all levels of illumination. Prolongation of the work to four hours increased the drop to 3.1 flashes/sec.[10]

It seems that the drop of the fusion frequency of flicker is more related to the general nature of the work performance and general fatigue of the central nervous system than to the visual component as such. Ryan, Bitterman, and Cottrell[11] arrived at a similar conclusion; they found no significant change of the fusion frequency of flicker in three and one-half hours of reading and a significant drop in mental calculations.

Schmidtke[5] found the same decrease of about six flashes in mental (blindfolded) or written calculations. The visual component (and, in the case of reading, also the mental component) is quite small in these series. Schmidtke's results show that a significant drop of the fusion frequency of flicker occurs also after complete elimination of the visual component.

The small, if not negligible role of the visual component is also shown by Ryan's, et al.,[11] finding that severe visual discomfort produced by glare did not affect the fusion frequency of flicker in three and one-half hours of reading. The assumption that the drop of fusion frequency of flicker during sedentary work is related essentially to the state of the central nervous system appears to be well supported.

Table 1 summarizes the changes observed by several authors in a variety of light occupational tasks. The scores of different authors are not directly comparable due to differences in the testing conditions. The slight difference of 1.3 flashes after 10 hours of

TABLE 1

CHANGES IN THE FLICKER FUSION FREQUENCY IN SEDENTARY WORK

Type of Work	No. of Subjects	Hours	Mean Change	Authors
Labor, clerical	17	8	−5.4	Simonson, et al.[3]
Laboratory	4	8	−4.6	Simonson, et al.[3]
Laundry	3	8	−1.6	Simonson, et al.[4]
Clerical	42	8	−0.4	Brozek[12]
Laboratory	26	8	−0.1	Brozek[12]
Mental calculations	8	1	−6.6	Arnold[13]
Mental calculations	5	3	−5.2	Schmidtke[5]
Microscopy	10	3	−5.3	Schmidtke[5]
Reading	12	3.5	−0.9	Ryan, et al.[11]
Reading	15	3	−0.9	Schmidtke[5]
Not stated	47	4	−2.4	Steinhaus & Kelso[14]
Driving	528	10	−1.6	Lee[15]
Linotype	3	8	−7.0	Schmidtke[16]
Fine assembly	15	8	−5.4	Schmidtke[16]
Pilot instructors	32	8	0.0	Graybiel[17]

driving was statistically significant.[15] The slight decrease of 1.6 flashes in laundry workers contrasts with the much larger drop in laboratory and clerical workers, tested with the same method.

Graybiel, et al.,[17] did not find any consistent change of the fusion frequency in pilot instructors during the working day. No high altitude flying was involved. Steinhaus and Kelso[14] reported a mean decrease of 2.4 flashes/sec. in 47 men from morning to noon. The occupation was not stated, but the subjects were probably college students or department employees. In contrast, Brozek, et al.,[12] found only very slight decrement of the mean fusion frequency of flicker in larger groups of clerical and laboratory workers after eight hours of work which reached the level of statistical significance in only one out of four comparisons.

The absence of a significant drop in reading[5, 11] is not surprising; the mental and visual component in ordinary reading is not large enough to produce fatigue.

The experience of the various authors is not uniform, but the majority found a significant drop during prolonged sedentary work. The variety of testing conditions may have contributed to the discrepancy of results.

The detailed study of Schmidtke[5] is of particular interest, since work performance (calculations) and change of the fusion frequency of flicker were compared in frequent intervals during three hours of work. We calculated means and standard deviations from Schmidtke's data, as shown in Table 2. The fusion frequency of flicker drops continuously from the first to the last period, while performance reaches a maximum at 60 to 90 minutes, and drops below the initial performance level only in the last period. The initial rise of performance, which is a known phenomenon, is not reflected in the fusion frequency of flicker.

Busch and Wachholder,[18] however, found

TABLE 2

PERFORMANCE (NUMBER OF ADDITIONS) AND FLICKER FUSION FREQUENCY IN 30-MINUTE INTERVALS OF CONTINUED WORK; MEANS AND S. D. OF 20 SUBJECTS SCHMIDTKE[5]

Time	0	30′	60′	90′	120′	150′	180′
Performance (means)	—	1307	1440	1442	1384	1322	1236
Standard deviation	—	311	314	318	316	288	269
Flicker fusion frequency (means)	49.5	47.4	46.0	45.0	44.3	43.7	42.7
Standard deviation	2.2	2.4	2.4	2.4	2.5	2.5	2.6

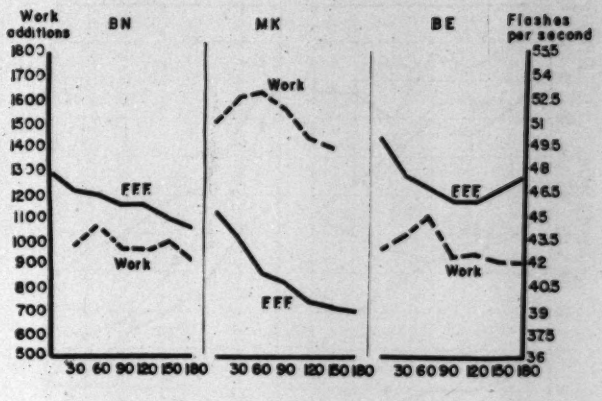

Fig. 1 (Simonson). Performance (number of additions, left ordinate) and flicker fusion frequency (right ordinate) during three hours of work, in three subjects. (From Schmidtke.[5] Figure 8.)

in the same type of performance an initial increase also in the fusion frequency of flicker, but much earlier (usually in the first five minutes, although occasionally later).

Individual discrepancies between changes of performance and of fusion frequency of flicker are shown in Schmidtke's series (fig. 1). In subjects B. N. and M. K., the decline after the performance peak at 60 minutes is parallel. Subject B. E. shows a terminal increase of the fusion frequency of flicker, while the work performance stays practically constant for 120 minutes. From the low steady performance level it was suggested that the subject did not exert himself in the last hour of the task, so that the terminal increase of the fusion frequency of flicker may represent a partial recovery of the central nervous system. In general, however, the drop of the fusion frequency of flicker was continuous, possibly representing the exhaustion of an energetic reserve.

This was disputed by Busch and Wachholder,[18] who found fluctuations in the drop of the fusion frequency of flicker during the same type of work (calculations), and missed also a parallelism to the subjective difficulty of the task. They found, however, a distinct drop of the fusion frequency of flicker, though in different subjects at different time of the work performance.

Another important approach to study of the relationship between fusion frequency of flicker and fatigue is investigation of work interruptions. The beneficial effect of rest pauses on fatigue is documented in a large literature (reviewed in part by Simonson.[19]) In three linotypists, the fusion frequency of flicker dropped consistently from the first hour to the lunch recess, increased during the pause without attaining the initial values, and continued to drop after the lunch pause (Schmidtke[16]). The drop of the fusion frequency of flicker and increase of errors in calculations were reduced and maintenance of performance improved by introduction of brief rest pauses at 30-minute intervals (fig. 2), and similar results were obtained in 15 workers engaged in fine assembly on a conveyor belt (Schmidtke[16]). In mental calculations caffeine delayed the drop of the fusion frequency of flicker (fig. 3), together with an improvement of the work performance.[5] The effect of rest pauses and caffeine agrees well with the concept that the fusion frequency of flicker is related to the central nervous system fatigue.

The effect of sleep deprivation is controversial; no significant change was found by Tyler[20] and by Brozek, et al.,[12] while Simonson and Enzer,[2] Riddell,[21] and Schmidtke[5] found a decrease of about −4.0 flashes. We found in 13 subjects who reported to work with common cold of light to moderate severity the fusion frequency of flicker consistently depressed, on the average by −3.7

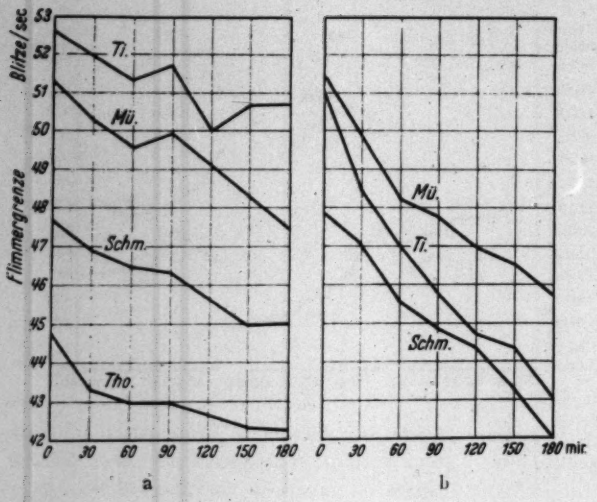

Fig. 2 (Simonson). Drop of flicker fusion frequency (ordinate) in work with (left) and without (right) pauses. (From Schmidtke.[8] Figure 9.)

flashes/sec., substantiating subjective discomfort and reduced subjective work capacity.

It is of interest that observation of the effect of some other stress situations are more uniform than the effect of fatigue, possibly because the element of voluntary cooperation is largely reduced. Therefore, results in other stress situations are pertinent for the appraisal of the general biologic sensitivity of the fusion frequency of flicker.

Figure 4 shows the effect of hypoxia (14 percent O_2) and acidosis (five percent CO_2 — 95 percent O_2) Simonson and Winchell[22]). Local acidosis may well be involved in the depression of the fusion frequency of flicker in central nervous system fatigue. We found the fusion frequency of flicker significantly lower in patients with cardiovascular disease, due to clinically latent cerebral ischemia.[23, 24] A relative, though slight, cerebral ischemia may be responsible for the lower fusion frequency of flicker in sitting than in supine position.[24] There is a surprisingly close parallelism between the decrease of the cerebral blood flow in Kety's[25] material and the drop of the fusion frequency of flicker with age, calculated as average of the results of various authors[26] (table 3).

Use of the fusion frequency of flicker is not suggested as criterion for physical fatigue, since more direct methods are available, but its response is of interest because of the large metabolic and circulatory changes in physical work. Interestingly enough, different types of physical work had a different effect on the fusion frequency of flicker (Simonson, et al.[27]). Static work producing fatigue within a few minutes increased the fusion frequency of flicker, probably due to

TABLE 3

CHANGES OF CEREBRAL BLOOD FLOW AND FLICKER FUSION FREQUENCY

Function	Average at Age (yr)	Decrease at Age (yr.)				
	20	30	40	50	60	70
Cerebral blood flow	60.0	−6	−10	−12	−14	−16
Flicker fusion	46.0	−1.3	− 2.8	− 4.5	− 5.8	− 9.0

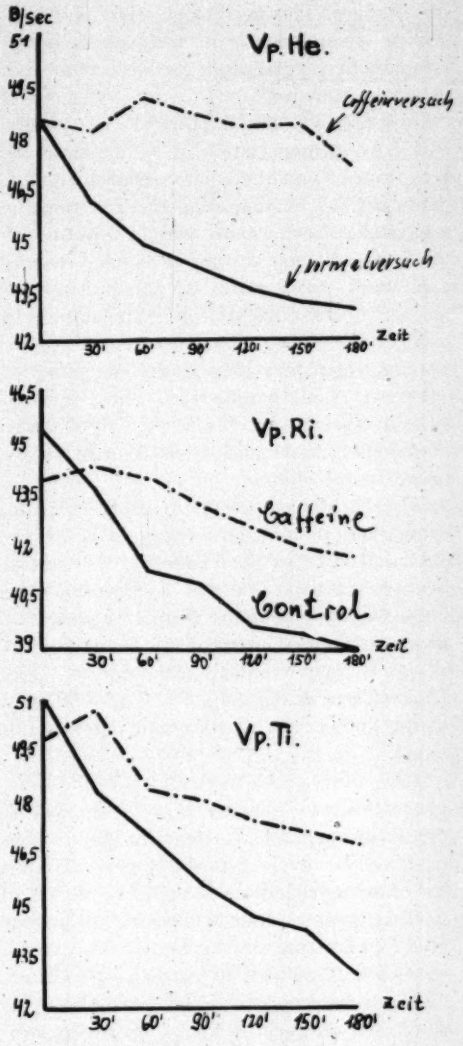

Fig. 3 (Simonson). Flicker fusion frequency during mental work (calculations) without (solid line) and with caffeine (broken line), in three subjects. (From Schmidtke.[5] Figure 12.)

intracentral spreading of excitation from the motor centers. This was confirmed by Schmidtke[5] and by Arnold and Wachholder[28] who accepted this interpretation.

Running to exhaustion decreased the fusion frequency of flicker, probably due to acidosis and hypoxia. Short, moderately heavy work produced two to four phasic fluctuations, interpreted as due to coexistence of stimulating and depressing factors. A triphasic response was confirmed by Arnold and Wachholder,[28] and interpreted as sympathetic-parasympathetic imbalance. Both interpretations are not mutually exclusive. Arnold[13] found a decrease of the fusion frequency of flicker in various types of exercise, followed by an increase after five to 10 minutes of recovery. Prolonged, moderate work at a steady-state (walking in the treadmill) did not change significantly the fusion frequency of flicker in Brozek and Keys'[29] experiments.

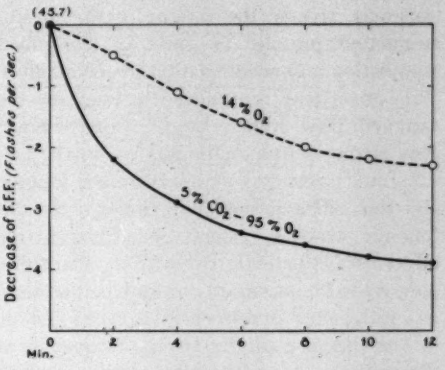

Fig. 4 (Simonson). Effect of 14-percent O_2 and of a mixture of five-percent CO_2 and 95 percent O_2 on the Means of 13 subjects. (From Simonson and Winchell.[22] Figure 1.)

In summary, physical work produces a variety of changes in the fusion frequency of flicker, dependent on type, severity, and duration of exercise, and probably involving some central factors which have still to be explored. The changes of the fusion frequency of flicker in the majority of experiments, however, revealed changes in the state of the central nervous system during physical work not previously detected.

One of the most important results in fatigue research of the last three decades is Orbeli's discovery that sympathetic stimula-

tion may reverse the fatigue of the in situ stimulated muscle, in spite of continued stimulation and accumulation of metabolites. This effect was reproduced in man for the fatigued pupil reflex by O. Lowenstein.[30] Any strong sensory stimulus (acoustic, tactile, optic) reverses pupillary reflex fatigue, and this effect is mediated through the autonomic nervous system. Sensitivity to the effect of sympathetic stimulation, therefore, appears to be important for any method used as a fatigue test of the central nervous system.

The increase of the fusion frequency of flicker after amphetamine[3] and pervitin[4] shows the sensitivity to sympathetic stimulation; these drugs stimulate the sympathetic centers with comparatively slight effect on peripheral circulation. Already early in our work we noticed often an increase of the fusion frequency of flicker to various acoustic or visual accidental stimuli, such as slamming a door, a person entering the room, and so forth. However, the effect of auditory stimuli was quite variable. Caloric vestibulary stimulation produced a highly significant decrease of the fusion frequency of flicker in normal persons and a significant increase in patients with the postconcussion syndrome.[31] Steinhaus and Kelso[14] found in 47 men a pronounced increase of the fusion frequency of flicker (6.4 flashes/sec.) on days with cold hip baths, compared to a decline by 2.4 flashes/sec. from morning to noon in control experiments. Most likely, the autonomic nervous system is involved in these reflex responses.

Schmidtke noticed a parallelism between diurnal fluctuations of the fusion frequency of flicker and adrenaline concentration in blood. The effect of the autonomic nervous system was thoroughly studied by Wachholder and Arnold.[32] Subcutaneous injection of adrenaline or suprarenine increased, and of Neurotropan, a parasympathetic stimulating drug, decreased the fusion frequency of flicker. The small doses used did not change the pupil. The effect increased with larger doses which also produced secondary opposite changes. This oscillatory effect is a well known phenomenon of sympathetic-parasympathetic regulation in cardiovascular and metabolic functions.

Lowenstein's pupillography[30] is ideally suited for demonstration of sympathetic effects, since sympathetic and parasympathetic pathways are anatomically and functionally separated. Lowenstein and Loewenfeld[33] used this method during prolonged sedentary work. In a series of successive light flashes, fluctuations of moderate amplitude occur before the work, demonstrating the normal, regulatory play in the sympathetic-parasympathetic equilibrium. These fluctuations are greatly increased after eight hours of sedentary work and are extreme in a state of profound fatigue.

The disturbance of the sympathetic-parasympathetic equilibrium seems to be an essential feature of the fatigue of the central nervous system. On this background, the large fluctuations of the fusion frequency of flicker at the end of the work task (calculations) in Busch and Wachholder's[18] series is most interesting (fig. 5). The similarity to the fluctuations of the pupil reflex is obvious.

Most likely, both methods show the same phenomenon: Disturbance of the sympathetic-parasympathetic equilibrium, which may well be involved in the greater variability of performance in fatigue, as shown in various types of industrial work and in our studies of visual performance.[9] This, however, is still subject to further exploration.

The sensitivity of the fusion frequency of flicker to changes of the sympathetic-parasympathetic equilibrium is a valuable feature but may obscure the drop of the fusion frequency of flicker. Wachholder and Arnold[32] suggest discarding of the fusion frequency of flicker for this reason. I feel, on the contrary, that the recent results, including those of Wachholder and his associates, open new perspectives in the application of the fusion frequency of flicker, which may ultimately lead to a better knowledge of the

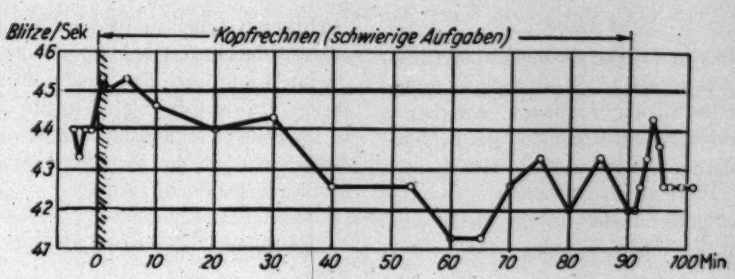

Fig. 5 (Simonson). Flicker fusion frequency before, during, and after 90 minutes of difficult mental work. The work period is marked by arrows. (From Busch and Wachholder.[38] Figure 3.)

central nervous processes in fatigue. A modification of procedure and interpretation, however, is necessary. Not only the absolute drop of the fusion frequency of flicker in longer time intervals, but also the fluctuations in shorter time intervals, particularly at the end of the work, should be evaluated. I wish to emphasize again that the fusion frequency of flicker is related to the excitability of the central nervous system and is not specific for fatigue. Concomitant factors, which might affect the central nervous system excitability, should be considered in the evaluation.

In the complexity of the situation, no single method could possibly be expected to be an adequate fatigue test for all types and conditions of work involving primarily the central nervous system.

If an independent, reliable method either for future changes or for changes in the autonomic nervous system was available, the interpretation would be reasonably safe. Since Lowenstein's pupillography appears to be a reliable index for changes of the sympathetic-parasympathetic equilibrium, the combined application of fusion frequency of flicker and pupillography holds real promise, the more so as no other objective method is yet available for central nervous system fatigue.

Exposure to flickering light produces a pronounced decrease of the fusion frequency of flicker.[31] These experiments were repeated and confirmed by Brozek and Simonson (unpublished data). Exposure to coarse flicker at a rate of 10 flashes/sec. below the fusion frequency produced a highly significant drop of 5.4 flashes/sec. after two minutes of exposure, and of 7.0 flashes/sec. after 10 minutes of exposure; these are average values for 10 normal subjects.

Exposure to fine flicker, at a rate of five flashes/sec. below the fusion frequency of flicker, produced an average of 2.8 flashes/sec. after 10 minutes, while exposure to rate of five flashes/sec. above the fusion frequency of flicker, that is, at subjectively steady light, did not change the fusion frequency of flicker. Similar results were obtained recently by Arnold.[4]

Exposure to very coarse flicker produced in 14 subjects a drop of seven flashes/sec. in two minutes, approaching a plateau after eight minutes.

The short duration of exposure seems to exclude "general" fatigue of the central nervous system as a basis for this phenomenon, the more so as the greatest drop occurs in the first minutes of exposure. It is probable that synchronization of brain potentials with the rate of flicker plays an important role in this phenomenon; a nervous center tuned to a low frequency might be temporarily impaired to follow a higher frequency. If this interpretation is correct, the described effect of flickering light is an adaptation phenomenon. However, it might be interesting to use this phenomenon in fatigue studies.

Summary

The effect of fatigue on the flicker fusion frequency is reviewed. Most authors found a drop of the fusion frequency of flicker in sedentary work, which can be counteracted by stimulating drugs and by rest pauses. However, there is no consistent correlation to general performance, to the visual component of performance, and to subjective fatigue.

Different types of physical exertion produce different effects on the fusion frequency of flicker. The fusion frequency of flicker is probably related to the excitability of the central nervous system, involving also disturbance of the sympathetic-parasympathetic regulation in fatigue.

Laboratory of Physiological Hygiene. (14).

References

1. Winchell, P., and Simonson, E.: Effect of light:dark ratio on the fusion frequency of flicker. J. Applied Physiol., **4**:188-192, 1951.
2. Simonson, E., and Enzer, N.: Measurements of fusion frequency of flicker as a test of fatigue of the central nervous system; observations on laboratory technicians and office workers. J. Indust. Hyg. & Toxicol., **23**:83-89, 1941.
3. Simonson, E., Enzer, N., and Blankstein, S. S.: Effect of amphetamine (benzedrine) on fatigue of the central nervous system. War Med., **1**:690-695, 1941.
4. Simonson, E., and Enzer, N.: Effect of pervitin (desoxyephedrine) on fatigue of the central nervous system. J. Indust. Hyg. & Toxicol., **24**:205-209, 1942.
5. Schmidtke, H.: Ueber die Messung der psychischen Ermüdung mit Hilfe des Flimmer-tests. Psycholog. Forsch., **23**:409, 1951.
6. Wachholder, K., and Schneider, W.: Die Anderung der Flimmer-verschmelzungsgrenze auf caffeinhaltigen Kaffee. Klin. Woch., **34**:236, 1956.
7. Larson, P. S., Finnegan, J. K., and Haag, H. B.: Effect of cigarette smoking on the fusion frequency of flicker. J. Clin. Investig., **29**:483-485, 1950.
8. Enzer, N., Simonson, E., and Ballard, G.: Effect of small doses of alcohol on the central nervous system. Am. J. Clin. Pathol., **14**:333-341, 1944.
9. Simonson, E., and Brozek, J.: Effects of illumination level on visual performance and fatigue. J. Optic. Soc. Am., **38**:384-397, 1948.
10. Brozek, J., Simonson, E., and Keys, A.: Changes in performance and in ocular functions resulting from strenuous visual inspection. Am. J. Psychol., **63**:51-66, 1950.
11. Ryan, T. A., Bitterman, M. E., and Cottrell, C. L.: Relation of critical fusion frequency to fatigue in reading. Illumin. Engin., **48**:385, 1953.
12. Brozek, J., Simonson, E., and Taylor, H. L.: Changes in flicker fusion frequency under stress. J. Applied Physiol., **5**:330, 1953.
13. Arnold, H.: Optische Verschmelzungsfrequenz und Ermüdung. Arbeitsphysiol., **15**:62, 1953.
14. Steinhaus, A. H., and Kelso, A.: Improvement of visual and other functions by cold hip baths. War Med., **4**:610-617, 1943.
15. Lee, R. H.: Fatigue and hours of service of interstate truck drivers. Critical fusion frequency of flicker. Pub. Health Bull. No. 265, 1941, pp. 195-208.
16. Schmidtke, H.: Ermüdungs-und Pausenprobleme bei körperlichen leichten und mittelschweren Arbeiten. Arbeitswiss., **5**:129-133, 1951.
17. Graybiel, A., Lilienthal, J. L., Jr., and Horwitz, D.: Flicker fusion tests as a measure of fatigue in aviators. J. Aviation Med., **14**:356-359, 1943.
18. Busch, G., and Wachholder, D.: Der Einfluss ermüdender geistiger Beanspruchung auf die Flimmerverschmelzungsfrequenz. Arbeitsphysiol., **15**:149, 1953.
19. Simonson, E.: Outline analysis of research problems in industrial physiology. Occupational Med., **3**:109, 1947.
20. Tyler, D. B.: The "fatigue" of prolonged wakefulness. Fed. Proc., **6**:218, 1947.
21. Riddell, L. A.: Use of the flicker phenomenon in the investigation of the field of vision. Brit. J. Ophth., **20**:380-418, 1936.
22. Simonson, E., and Winchell, P.: Effect of high carbon dioxide and of low oxygen concentration on fusion frequency of flicker. J. Applied Physiol., **3**:637-641, 1951.
23. Enzer, N., Simonson, E., and Blankstein, S. S.: Fatigue of patients with circulatory insufficiency, investigated by means of fusion frequency of flicker. Ann. Int. Med., **16**:701-707, 1942.
24. Keys, A., and Simonson, E.: Flicker fusion frequency and its response to nitroglycerin in normal subjects and in patients with cardiovascular disease. Circulation, **5**:215-224, 1952.
25. Kety, S. S.: Changes in cerebral circulation and oxygen consumption which accompany matura-

tion and aging. In Biochemistry of the Developing Nervous System (H. Waelsch). New York, Academic, 1955, p. 208.

26. Simonson, E.: Changes of physical fitness and cardiovascular functions with age. Geriatrics, **12**: 28-39 (Jan.) 1957.

27. Simonson, E., Enzer, N., and Benton, R. W.: Influence of muscular work and fatigue on the state of the central nervous system. J. Lab. & Clin. Med., **28**:1555-1567, 1943.

28. Arnold, H., and Wachholder, K.: Weitere Untersuchungen über optische Verschmelzungsfrequenz und ermudende körperliche Beanspruchung Arbeitsphysiol., **15**:139, 1953.

29. Brozek, J., and Keys, A.: Flicker fusion frequency as a test of fatigue. J. Indust. Hyg. & Toxicol., **26**:169-174, 1944.

30. Lowenstein, O.: Der psychische Restitutionseffekt. Das Prinzip der psychisch bedingten Wiederherstellung der ermudeten, der erschöpften und der erkrankten Funktion. Basel, B. Schwabe, 1937.

31. Simonson, E., Fox, M. S., and Enzer, N.: Influence of vestibular stimulation on the fusion frequency of flicker in normal subjects and in patients with postconcussion syndrome. Arch. Otolaryng., **38**:245-251, 1943.

32. Wachholder, K., and Arnold, H.: Ueber die vegetative Steuerung der Leistungsfahigkeit unseres Lichtsinnesapparates gemessen an der Flimmerverschmeizungsfrequenz, Ztschr. f. Biol., **107**:252, 1954.

33. Lowenstein, O., and Loewenfeld: I. Disintegration of central autonomic regulation during fatigue and its reintegration by psychosensory controlling mechanisms. J. Nerv. & Ment. Dis., **115**:121, 1952.

34. Snell, P. A.: An introduction to the experimental study of visual fatigue. J. Soc. Motion Picture Engineers, **20**:367-390, 1933.

ON THE ETIOLOGY, PATHOLOGY, AND SURGICAL TREATMENT OF RETINAL DETACHMENT*

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I. THE DEVELOPMENT OF THE SCLEROTIC AREAS IN THE RETINA

The importance of the vitreous in the pathogenesis of retinal detachment has been recognized for about 100 years. Primary retinal detachment is always accompanied by vitreous detachment. De Wecker and Leber long ago noted the significance of retinal holes as a cause of detachments. Clinical and experimental experiences by Gonin and Lindner pointed to the traction of the vitreous body on the retina and attributed this to inflammation. Unfortunately evidence of such inflammation is rarely found.

Vogt thought that retinal holes were caused by decay in the retina itself—that is, "cystoid degeneration." Against this view it may be said that holes are usually situated near vessels. The vessels are rarely obliterated, and distal to the sclerotic area they appear of normal caliber.

This paper will concern itself with the nature of the attachment of the vitreous and the retina, the histologic findings in the sclerotic areas predisposing to retinal breaks, and finally a method of surgical treatment for retinal detachment.

Most authors consider cystoid degeneration (Blessig-Iwanoff's edema) to be the beginning of retinal holes. This is not the case, as will be shown. The clinical picture of the areas predisposing to retinal breaks is well known. These areas are usually situated between the equator and the ora serrata and are not seen frequently. Contrarily the cystoid areas found in histologic preparations are very frequent. In these cases the retina is ruptured in the bipolar cell layer, and yet there is no surrounding tissue reaction (fig. 1). It is primarily the structure of the vitreous attaching to the retina at the posterior face which ruptures the retina from its attach-

* From the University Eye Clinic. This paper was presented before a group of ophthalmologists at Havana, Cuba, on June 17, 1958, and at a meeting of the Retina Foundation at Ipswich, Massachusetts, May 30, 1958.

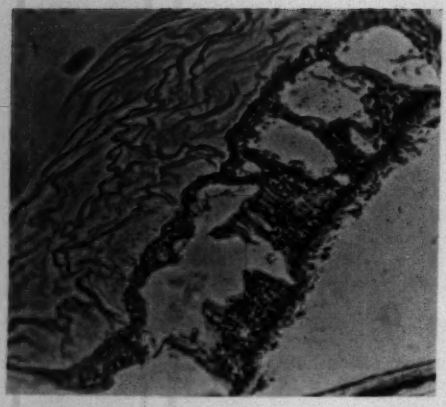

Fig. 1 (Pau). "Cystoid degenerations" usually are ruptures of the retina by shrinkage (in histologic preparation) of the structure of the vitreous attaching to the retina at the posterior vitreous face. (Phase contrast 1:100.)

ment by shrinkage during histologic fixation (fig. 1). Such cystoid degenerations are not found during life.

In order to understand the attachment of the vitreous to the retina, we must regard the normal structure of the vitreous. This structure corresponds to the embryonal blood vessel pattern (fig. 2). Note the vessels (a-f) which contribute to the structure of the vitreous, connecting the retinal vessels in the equator and the tunica vasculosa lentis.

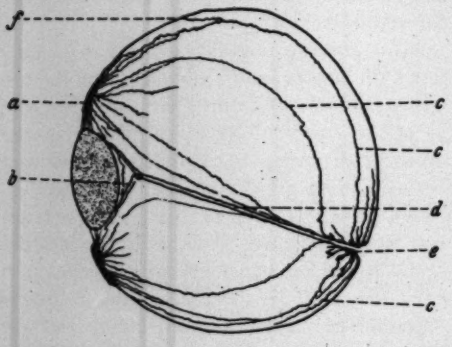

Fig. 2 (Pau). The structure of the vitreous (Ox) corresponding to embryonal blood vessels. (Connection structure of vitreous-retina = f.) (Arch. f. Ophth., **152**:201, 1951.)

Fig. 3 (Pau). The connection between the structure of the vitreous and the retina. (1:25.)

One of the later physiologic attachments between vitreous and retina is seen in Figure 3. If the vitreous body then becomes fluid, the movable structure of the vitreous draws on the attachment of the retina and, if the retina is damaged, a retinal hole results.

Sometimes the retina itself shows various changes at the point of vitreous attachment. Here retinal tissue is replaced by collagenous connective tissue and we see it hyalinized and fibrosed as in Figure 4. Frequently attachment of the vitreous to hyalinized blood vessels is seen (fig. 7). The lamina vitrea of the choroid is thickened in these areas also (fig. 5). The connection between the structure of the vitreous and the retina may appear as a cord or as a curtain. Usually the histologic picture is one of double curtains (figs. 3 and 5).

The sclerotic areas are not innate. Perhaps their beginning is best explained by the mechanical damage to the retina caused by the vitreous pull at its point of attachment. The newly formed perivascular connective tissue

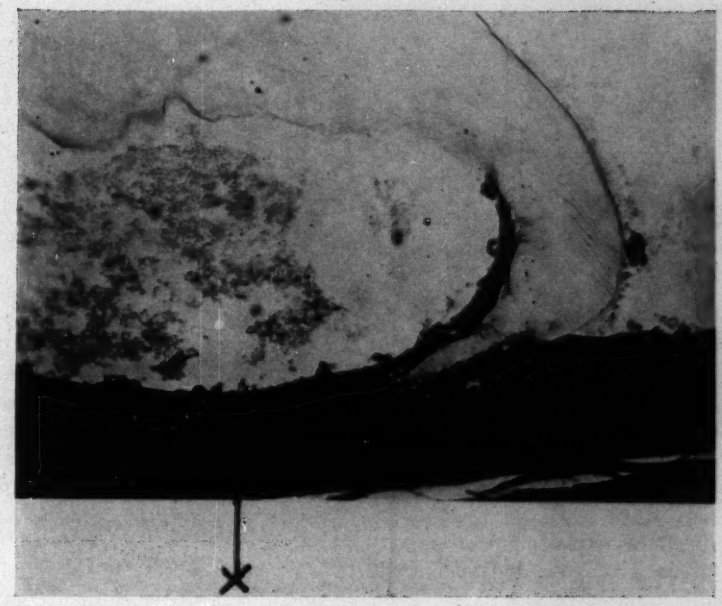

Fig. 4 (Pau). New connective tissue and the pigment epithelium of the retina growing over the structure of the vitreous. Sclerotic area (x). (1:200.)

and the retinal pigment epithelium show the reaction of fibrosis and hyalinization already mentioned, resulting in the "sclerotic areas."

In summary: Sclerotic areas predisposing to retinal detachment begin where the embryonal blood vessels of the vitreous communicate with the retinal vessels. In these areas the retina is supplanted by connective

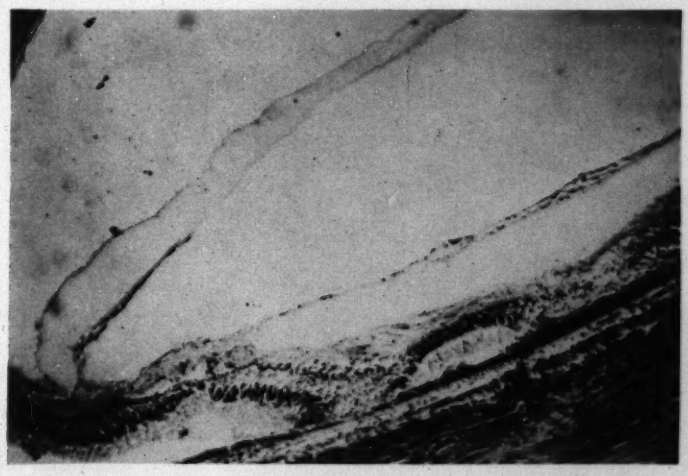

Fig. 5 (Pau). Attachment of the structure of the vitreous and the retina. The new connective tissue and the pigment epithelium of the retina grow over the structure of the vitreous. (1:120.)

Fig. 6 (Pau). A break of the retina caused by the attached structure of the vitreous. (Zwangl. Abh. aus dem Gebiet der Augenh. Heft 13.) (1:180.)

tissue and modified retinal pigment epithelium. The tissue is sclerotic and hyalinized and susceptible to retinal breaks.

II. Retinal breaks

Retinal holes are usually not situated in the sclerotic areas themselves. In spite of their thinness, these areas are very cohesive. For example, in horseshoe breaks, the sclerotic areas are mostly found in the operculum or flap. Retinal rupture begins primarily at the junction of sclerosed and normal retina, where rarefaction of cells and loosening of retinal tissue occur. Figures 6 through 10 demonstrate different cuts through a retinal hole. In the center of the hole, the sclerosed area is drawn out of the retina by a vitreous strand (fig. 6). Peripherally in the hole the rupture lies in the normal retina (figs. 7 and 8), and further peripheral to this, the inner layers of the retina are preserved (fig. 9). The most peripheral cut in the hole shows a rupture of only the deeper layers of the retina (fig. 10). As already mentioned, the vitreous is attached to the peripheral retinal vessels. It is not surprising, therefore, that retinal vessels can rupture and result in vitreous hemorrhages.*

III. A technique of retinal detachment surgery

Gonin succeeded in closing retinal holes by cicatrization of the sclera, choroid, and retina by a Paquelin. Weve used ball diathermy, and either this method or diathermic needles

* Fully illustrated details of the histologic findings in such cases are given in my book *Reaktive Zellveranderungen in Hornhaut und Netzhaut,* Verlag Carl Marhold, Halle, 1957.

Fig. 7 (Pau). Laterally from Figure 6 the rupture lies on the normal retina. The structure of the vitreous attaches to blood vessels. Sclerotic area (x). (Zwangl. Abh. aus dem Gebiet der Augenh. Heft 13.) (1:200.)

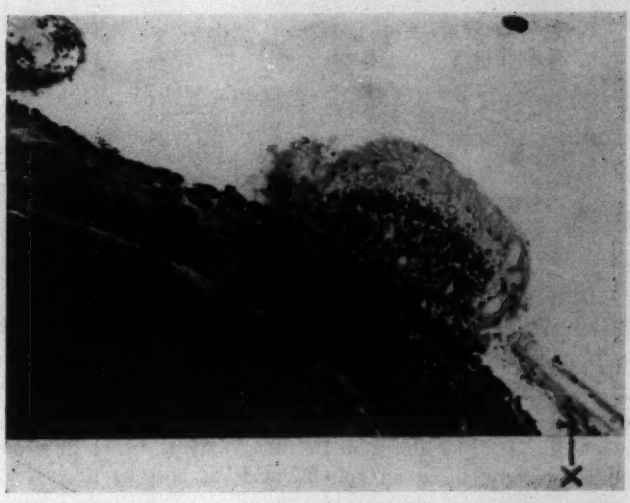

Fig. 8 (Pau). Laterally from Figure 7 the attaching structure of vitreous is seen. Sclerotic area (x). (Zwangl. Abh. aus dem Gebiet der Augenh. Heft 13.) (1:200.)

are usually used now. Lindner (after L. Müller) was the first to shorten the globe. Rosengren injected air into the globe. Weve introduced reefing of the sclera. Lamellar scleral resection is often performed now. In 1949, Custodis began sewing a "plombe" (tube) onto the sclera. As a disciple of Custodis, I have been using his technique since that time.

METHOD*

Patients are operated upon the day after admission. The sclera is exposed (fig. 11) after a small conjunctival incision, and without canthotomy or detaching the muscles. Exact localization of the holes is done with

* Covered extensively in "Die Operation der Netzhautablosung mit der Plombe." Ophthalmologica (Basel) 1958.

Fig. 9 (Pau). Laterally from Figure 8. The inner layers of retina are presented. (Zwangl. Abh. aus dem Gebiet der Augenh. Heft 13.) (1:200.)

Fig. 10 (Pau). Laterally from Figure 9. Only the deeper layers of the retina are ruptured. (Zwangl. Abh. aus dem Gebiet der Augenh. Heft 13.) (1:200.)

transillumination. (An Amsler marker is pressed on the sclera and its location noted by indirect ophthalmoscopy, using an ophthalmoscope with a downward decentered light, Figure 12.) Coagulative diathermy is performed on the sclera directly over the breaks (fig. 13).

Supramid sutures are placed in the sclera about two to three mm. from the borders of the hole (fig. 14), and then a "plombe" (tube) of polyviol is tied beneath the sutures, thereby invaginating the sclera (fig. 15). The "plombe" must overlie the hole exactly, either radially (fig. 16) or circularly (fig. 17). One or two 4.0-mm. diameter tubes may be used in order to make the buckle as deep as necessary. After placing the tube, one may see a large prominence in the fundus by indirect ophthalmoscopy (figs. 18 and 19).

If the retinal hole is not located on the

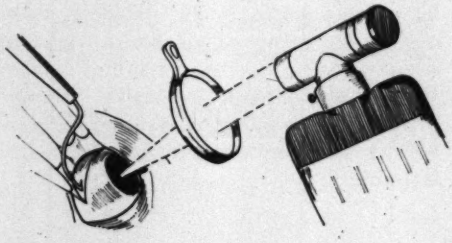

Fig. 12 (Pau). Transillumination with the downward decentered light and visualization of the marker by indirect ophthalmoscopy.

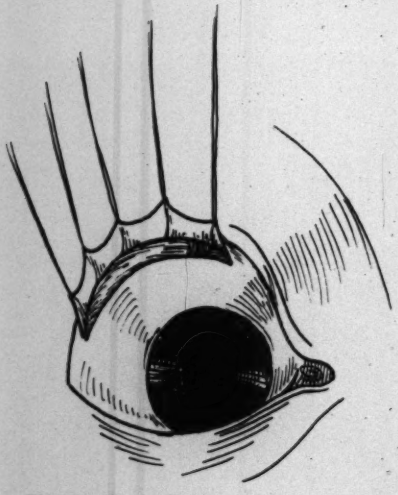

Fig. 11 (Pau). Exposure of the sclera.

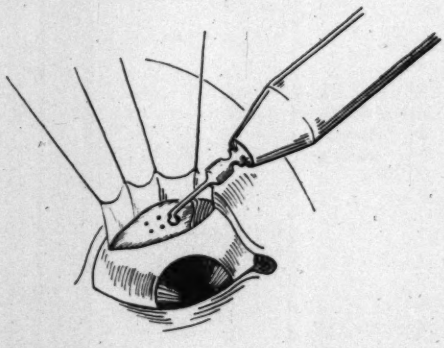

Fig. 13 (Pau). Coagulative diathermy on the sclera.

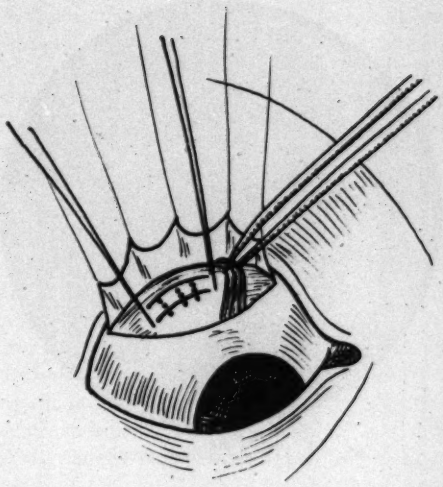

Fig. 14 (Pau). Two sutures of Supramid are tied on the sclera about two or three mm. from the border of the hole.

buckle but rather on the slope away from the buckle crest, the detachment will not heal. Usually the retina is reattached the next day. Subretinal fluid routinely reabsorbs without scleral perforation. Only in large detachments do we perforate to allow escape of subretinal fluid. Patients are ambulatory and

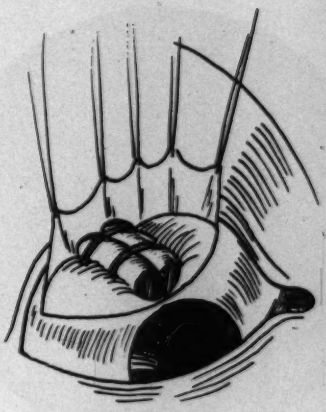

Fig. 16 (Pau). The tubes are fixed radially on the sclera with the sutures.

are given their pinhole glasses one or two days after surgery.

Results

During the last three years, 433 retinal detachments have been operated; 369 of these, or 85 percent, were healed (the retina was reattached). Of the 290 detachments with retinal breaks (excluding those with aphakia, perforating wounds, holes of the ora serrata, and periphlebitis), 262 or 90 per-

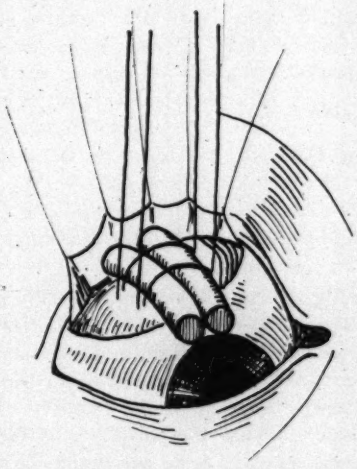

Fig. 15 (Pau). The tube or tubes of polyviol are placed under the sutures.

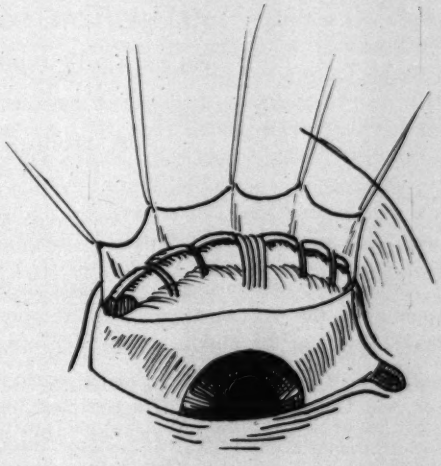

Fig. 17 (Pau). The tubes are fixed circularly on the sclera with the sutures.

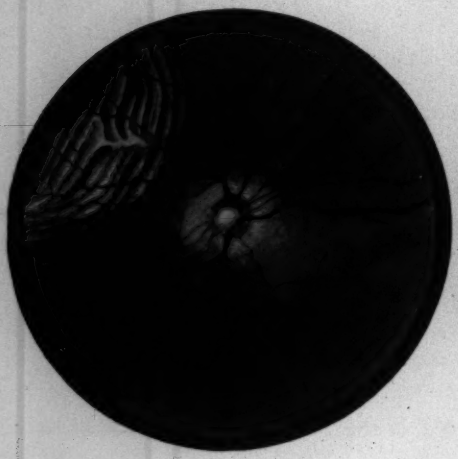

Fig. 18 (Pau). Tubes radially.

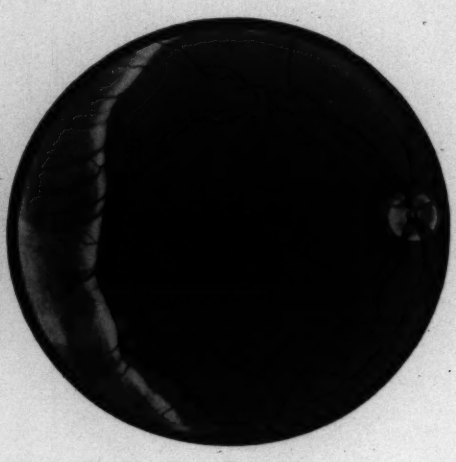

Fig. 19 (Pau). Tubes circularly.

cent were reattached. Breaks were found in 90 percent of our cases using the indirect ophthalmoscope with the downward decentered light.* A secondary procedure was necessary on 56 patients, and two patients required a third operation with a "plombe" (tube). Sixty-two percent of 24 detachments in aphakia (11 of these without breaks) were healed. Of 58 detachment with breaks at the ora serrata, 88 percent were reattached. Of 61 patient (11 aphakics, 10 perforated wounds), no hole could be found but nevertheless 41 (67 percent) were reattached using a tube.

University Eye Clinic.

* Klin. Monatsbl. f. Augenh., 129:691, 1956.

ASPHERICAL OBJECTIVE LENSES*

As an aid in indirect ophthalmoscopy

A preliminary report

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AND
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Indirect ophthalmoscopy was described soon after the first mention of direct ophthalmoscopy in the middle of the 19th century. By the end of that century Gullstrand had designed a reflex-free binocular indirect ophthalmoscope which utilized an aspherical objective.[1] The size and complexity of this instrument, as well as the inability of the examiner to view the periphery of the retina, prevented its wide acceptance for routine use.

Indirect ophthalmoscopy with a mirror

* From the Retina Service of the Manhattan Eye, Ear, and Throat Hospital and The Eye-Bank for Sight Restoration, Inc., New York, and the Department of Ophthalmology, School of Medicine, Western Reserve University, Cleveland. This study was aided by a grant from the Lillia Babbit Hyde Foundation.

and lens was popular and used both here and abroad about 40 years ago. The condensing lenses were spheres, varying between 10 and 14 diopters, which were usually obtained from the trial lens set. The difficulty in aligning the patient's eye, the condensing lens, and the examiner's eye, and the amount of time required to dilate the pupil caused many to abandon the technique. Following the development of the self-illuminated direct ophthalmoscope, indirect ophthalmoscopy was used less and less, and many of the American ophthalmologists did not use it at all. In Europe, however, the technique of indirect ophthalmoscopy remained popular. Further improvement in its use was obtained by using more intense light sources and larger mirrors, both factors improving the brightness of the image.

The objective lens used in indirect ophthalmoscopy performs two functions: condensing the light from the source toward the entrance pupil, thereby illuminating the fundus, and forming an inverted real image of the fundus. The magnification of the aerial image can be approximately determined by dividing the dioptric power of the eye by the dioptric power of the condensing lens. For example, if the dioptric power of the eye is assumed to be 60 diopters and that of the condensing lens, 15 diopters, then magnification equals 60/15 or 4.* Thus, the stronger the condensing lens, the smaller will be the aerial image.

About 10 years ago, Dr. Charles Schepens of the Massachusetts Eye and Ear Infirmary developed a self-illuminated binocular indirect ophthalmoscope. This instrument greatly increased the ophthalmologist's ability to examine the fundus, especially the peripheral portions. Although it requires training to master the use of this instrument, it is of considerable value and superior to direct ophthalmoscopy in the examination of retinopathies, retinal separation, retinal tumors, intraocular foreign bodies, and in the ability to see fundus lesions when there are opacities of the ocular media.[3,4]

The condensing lens which has been used in conjunction with the binocular indirect ophthalmoscope is a 20-diopter biconvex sphere with one fifth of its power on one surface and four fifths of its power on the surface facing the examiner. It is 32 mm. in diameter, coated to reduce reflections, and mounted in a black metal ring. Even with this lens of small diameter, the quality of the aerial image is poor because of the aberrations resulting from the use of spherical refracting surfaces. The use of higher powered spherical lenses or lenses of larger diameter is not practical since the aberrations become more pronounced with the increase in power or diameter. An attempt to overcome the aberrations of the single spherical lens by the use of spherical doublets has been unsatisfactory due to the internal reflections and the excessive thickness of the lens combinations.[5]

Recently, one of us (D. V.) has undertaken a systematic investigation of the optical properties of conoid lens surfaces and has produced a series of strong plus aspherical spectacle lenses used as reading aids in subnormal vision. These crown-glass lenses were designed to reduce or eliminate the aberrations which are present in spherical lenses, making them unsatisfactory in high powers.[6] It was felt that these lenses would offer an advantage over spherical lenses for indirect ophthalmoscopy. Accordingly, a series of available conoid lenses was employed for indirect ophthalmoscopy and compared to spherical lenses. The results with respect to the quality and size of the aerial image are presented in Table 1.

The 15-diopter lens is employed to obtain greater magnification of small lesions. The depth of focus is extremely shallow and as a result the lens must be held with a steady hand at a point almost exactly eight cm. from the emmetropic eye. The spherical lens, because of its aberrations, produces an im-

* Modified from the following (formula A):[2]

$$\text{Magnification equals } \frac{1{,}000}{\text{Dioptric power of lens} \times 15}$$

TABLE 1

SUMMARY OF EXPERIENCE WITH SPHERICAL AND CONOID LENSES IN BINOCULAR INDIRECT OPHTHALMOSCOPY

Power (in diopters)	Working Distance (in cm.)	Type of Lens Surfaces	Diameter (in mm.)	Magnification of Real Image	Degree of Definition	Peripheral Distortion of Aerial Image	Brilliance and Clarity	Depth of Focus (in mm.)	Degrees of Fundus in One Field
15	7.5 to 8.5	Plano convex sphere	50	×4.4	C	Marked	B	2 to 3	18–20
15	7.5 to 8.5	Plano convex conoid	50	×4.4	B	Slight	A	2 to 3	18–20
20	5 to 7	Biconvex sphere	31	×3.3	B	Slight	A	3 to 5	29–32
20	5 to 7	Plano convex conoid	30	×3.3	A	Almost none	A	3 to 5	29–32
20	5 to 7	Plano convex conoid	37	×3.3	A	Negligible	A	3 to 5	30–35
30	4 to 6.5	Biconvex sphero conoid	30	×2.2	B	Negligible	B	4 to 6	35–45
40	3.5 to 6.5	Biconvex sphero conoid	30	×1.7	B	Slight	C	5 to 7	45–55
60	3 to 5	Biconvex biconoid	30	×1.1	C	Moderate	D	7 to 10	65–90

EXPLANATIONS

The conoid lenses described here were corrected for a distance of 10 mm. from the eye. This distance is based on geometric construction modified by measurement under working conditions in a variety of refractive states.

The magnification is calculated from formula A and represents the magnification of the real aerial image in emmetropia. The magnification of the image formed by the conoid lenses is very slightly less over-all than the spherical lenses, since conoid lenses have a uniform magnification for the entire image, whereas the magnification of the spherical lenses changes peripherally.

Definition, clarity and brilliance of the images are graded empirically as follows: A—excellent; B—good; C—satisfactory; D—poor.

Peripheral distortion is empirically described.

Depth of focus is an estimate obtained from viewing elevated lesions under a variety of conditions.

The extent of the fundus viewed in one field varies with the refraction and the pupillary size and the figures given represent a combination of simple estimates with derivations from first-order geometric optical constructions.

age with marked peripheral blurring and distortion. Despite the fact that the aspherical lens used in the initial test was designed for placement one cm. from the cornea, the correction of aberrations was such that the image was noticeably free from blurring and distortion even when the lens was held eight cm. from the cornea, and it was possible to fill the whole diameter of the lens with a sharply focused image. With the spherical counterpart, only the central portion of the lens could be focused clearly.

The 20-diopter lens has a shorter working distance, permitting the examiner to steady the lens by resting his third and fourth fingers against the patient's forehead. The greater depth of focus permits greater facility in obtaining a full field. Distortion of the peripheral portions of the image was noticeable in the spherical lens and absent in the aspherical lens even though the aspherical lens was six-mm. greater in diameter than the spherical lens.

The 30-diopter aspherical lens is very easily focused and includes a larger area of the fundus in the image. Peripheral distortion remains negligible. The shorter working distance and greater depth of focus are particularly valuable to the beginner and in the operating room. There is a very slight haziness associated with the internal reflections. The 30-diopter lens, when used to examine the ora serrata, reduces the magnification, increases the illumination remarkably, and produces a more easily interpreted image. The larger area of the fundus in the image enables the surgeon to evaluate the distribution of subretinal fluid with ease, enabling him to perforate more accurately for better drainage. This high-powered lens reduces the magnification of the aerial image in high myopia and is an advantage in understanding fundus topography. In examining eyes with small, fixed pupils, that is, congenital cataracts after extraction, the larger field of the 30-diopter lens is of great value.

TABLE 2

CONOID LENSES DESIGNED FOR INDIRECT OPHTHALMOSCOPY*

Power (diopters)	Type	Diameter (in mm.)	Use Recommended
30	Biconvex sphero-conoid	31	Beginner; large field & increased depth of focus; for operating room; for high myopes; small, fixed pupils
20	Plano-convex-conoid	35	Routine use. Very clear image; large, undistorted field
15	Plano-convex-conoid	45	For posterior pole, disc & macula; for fine detail in small lesions

* Manufactured by the American Bifocal Co., Cleveland, Ohio.

The 40-diopter aspherical lens produces a still greater field of view and depth of focus. There is noticeable haziness of the image due to internal reflections.

The image produced by the 60-diopter conoid lens is less brilliant. The field of view is approximately 90 degrees. Magnification is approximately one. The use of this extremely strong lens for indirect ophthalmoscopy is at present recommended for experimental purposes only.

The aspherical lenses used in this study are a distinct improvement over available spherical lenses. We have now completed the development of a series of aspherical lenses especially designed and corrected for use in indirect ophthalmoscopy. Table 2 represents our recommendation as to the most useful powers and diameters of these lenses for this purpose.

SUMMARY

1. Although indirect ophthalmoscopy was described over a century ago, its use in the United States was virtually abandoned until the introduction of the binocular indirect ophthalmoscope 10 years ago.

2. Until the present time spherical lenses exclusively have been employed as the objective-condensing lenses for indirect ophthalmoscopy. The maximum power and diameter of these lenses was limited due to spherical aberration.

3. We have successfully employed existing conoid types of aspherical lenses for indirect ophthalmoscopy in powers and diameters equivalent to and greater than that of available spherical lenses. The aspherical lens appeared to be distinctly superior to its equivalent spherical lens under all conditions.

4. We have designed a series of conoid lenses especially corrected for indirect ophthalmoscopy and recommend the use of the 15, 20, and 30-diopter lenses for this purpose.

210 East 64th Street (21).
2065 Adalbert Road (6).

REFERENCES

1. Duke-Elder, W. S.: Textbook of Ophthalmology. St. Louis, Mosby, 1946, v. 1, pp. 785-805.
2. Ibid.: Page 804.
3. Schepens, C. L.: A binocular indirect ophthalmoscope. Tr. Am. Acad. Ophth., **51**:298, 1947.
4. Havener, W. H.: Schepens' ophthalmoscope. Am. J. Ophth., **45**:915, 1958.
5. Okamura, I.: Personal communication, 1956.
6. Volk, D.: Conoid refracting surfaces and conoid lenses. Am. J. Ophth., **46**:86 (July, Pt. II) 1958.

PREVENTION OF RETINAL DETACHMENT IN CATARACTOUS EYES

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When the retina detaches in an eye from which a cataractous lens has been removed, the same sequence of events may occur in the fellow eye. The frequency of such a complication can be reduced by attaching the peripheral retina firmly to the sclera by means of diathermy. This prophylactic measure is more simple and is more likely to result in retention of vision than are reparative techniques as scleral shortening and vitreous implant. Also, if the retina has detached in a noncataractous eye, cataract extraction upon the other eye may initiate changes which will detach the retina in it. Therefore, in such cases, prevention is also indicated.

Several months before the extraction of cataracts for such "second eyes," Franceschetti makes a prophylactic barrage in the two upper quadrants, which he believes is adequate. He has found that cauterization at 12 to 13 mm. from the limbus does not affect the limits of the visual field.

For the past five years, I have modified Franceschetti's technique and have applied nonpenetrating diathermy to the sclera completely around the globe just posterior to the ora serrata.

This procedure is also indicated prior to cataract extraction in eyes in which (1) idiopathic detachment has occurred in the opposite eye with or without cataract formation, (2) peripheral cystic degeneration of the retina has developed, and (3) in highly myopic (−20D.) eyes with evidence of choriodal or retinal stretching. It is not indicated when a retinal reattachment has been done on the cataractous eye in question, because such an eye already has strong sclerochoriodoretinal adhesions; in my experience, the retina has not detached in such an eye after cataract extraction.

The technique of the "retinal sealing" operation, or coagulation of the ora serrata, is as follows (fig. 1):

A circular incision through the conjunctiva and Tenon's capsule is made around the

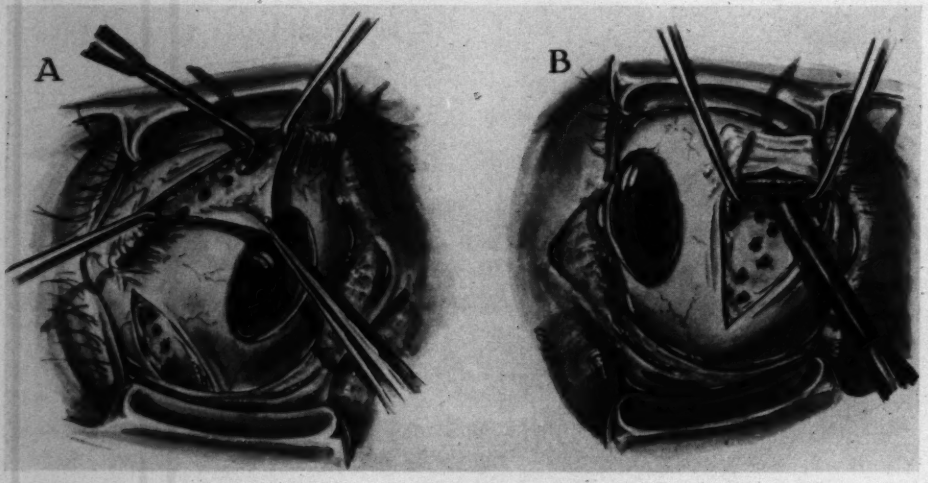

Fig. 1 (Callahan). Technique of retinal sealing operation.

TABLE 1

SURVEY OF EIGHT PATIENTS

Patient Sex Age (yr.)	Details of Retinal Detachment of First Eye	Date of Retinal Sealing Second Eye	Date and Type of Cataract Extraction Second Eye	Date of Last Observation and Corrected Vision
C. H. w.m. 24	(Left) Dec. '51 extracapsular extraction, fluid vitreous was present, none threatened loss. Oct. '53 retinopexy including scleral buckling but later complete re-detachment.	(Right) Apr. 12, '54	July 12, '54 Extracapsular, small loss vitreous.	Feb. '56 20/20
J. E. C. w.f. 73	(Right) Cataract was removed Dec. '53. When first examined by me in May '54, the retina had been detached for some months. Case complicated by severe senile cicatricial entropion which required surgical correction before surgery of the globe begun.	(Left) July 23, '54	Nov. 26, '54 Intracapsular, no complications.	Dec. '58 20/30
W. J. W. w.m. 82	(Right) Cataract extraction performed elsewhere in Apr. '53, followed by hemorrhage three days postoperatively, and when first examined by me seven months later massive detachment of retina present. Chronic uveitis required enucleation, Apr. '54.	(Left) Aug. 16, '54	Feb. 4, '55 Intracapsular, no complications.	July '55 20/20
D. A. R. w.m. 56	(Right) Cataract extraction elsewhere followed 3 months later by detachment after the operation, and two unsuccessful attempts at reattachment.	(Left) Sep. 16, '55	Dec. 28, '55 Intracapsular, no complications.	Apr. 29, '58 20/20
P. F. w.f. 55	(Left) Cataract was removed Nov. '54. When first examined by me in July '55, retina was completely detached.	(Right) Oct. 3, '55	Apr. 16, '55 Intracapsular, no complications.	July '58 20/40
M. J. w.m. 65	(Right) Extracapsular extraction of hypermature lens, Nov. '54, reattachment achieved Nov. '56, complication of corneal dystrophy after orbital cellulitis, visual acuity never regained better than 20/200.	(Left) Nov. 12, '56	Mar. 11, '57 Intracapsular, no complications.	Jan. 21, '58 20/20
W. M. Mc. w.f. 73	(Right) Idiopathic detachment, left eye, about '45. No surgical reattachment attempted.	(Left) Feb. 18, '57	May 10, '57 Intracapsular, fluid vitreous presented.	Feb. '59 20/25
E. C. Mc. w.f. 70	(Right) Retinal detachment after cataract operation performed in another city, then enucleation.	(Left) Jan. 16, '58	May 29, '58 Intracapsular, no complications.	Feb. '59 20/40

globe between the insertions of the rectus tendons, omitting severance of these two layers at the insertions themselves. These four uncut sections permit a more rapid return of normal metabolism to the limbal-based conjunctival ring than if it were completely severed from the remainder of the conjunctiva. The sclera is exposed by undermining Tenon's capsule between each rectus tendon insertion.

A. A line of diathermy contact points, each about two mm. apart, is made extending between the tendon insertions, at a distance of about 10 mm. from the limbus. The current

is applied gently—long enough at each site for the surface to take on a greenish tinge, but not long enough for the surface to turn brown. Any type of electrode may be used.

B. To create adhesions between the retina, choriod, and sclera beneath the insertions, diathemy current is applied to the sclera beneath the tendons close to the insertions with the Rychner electrode.

The conjunctival incisions are then closed with interrupted sutures (chromic 6-0). The extraction of the cataractous lens is performed three to four months later.

When the cataract is extracted, every effort should be made to avoid loss of vitreous or rupture of the capsule which might result in prolonged iridocyclitis. Either vitreous loss or prolonged iridocyclitis may result in the condensation of the vitreous sheets producing traction bands. Also, fibrous tissue may result from a migration of cells from the corneal wound or from accumulation of inflammatory cells on the condensed strands of vitreous. These may eventually contract to produce traction bands. If vitreous is lost at extraction, the wound should be freed of vitreous as much as possible, and the wound should be closed very tightly with four or five corneoscleral sutures (chromic 6-0). If the capsule is ruptured, despite the thoroughness of the removal of the lens material, cortone, antibiotics, and atropine should be liberally used postoperatively.

This procedure has now been used for eight patients (table 1), the earliest of which were operated upon four and one-half years ago. All of these patients have had a successful outcome with normal aphakic vision, though no doubt failures will occur when the numbers of these procedures have increased enough for the relentlessness of the law of averages to be imposed.

903 South 21st Street.

References

Callahan, A.: Surgery of the Eye Diseases. Springfield, Ill., Thomas, 1956.

Franceschetti, A.: New operations and some special indications and techniques in ophthalmic surgery. Am. J. Ophth., **39**:189-197 (Feb.) 1955.

Guyton, J. S.: Complications after cataract extraction: Pathologic aspects. In symposium: Cataract extraction. Tr. Am. Acad. Ophth., **58**:397-407 (May-June) 1954.

Maumenee, A. E.: Postoperative complications: Retinal detachment. Tr. Am. Acad. Ophth., **61**:51-68 (Jan.-Feb.) 1957.

Rychener, R. O.: Weve diathermy electrode: A new modification. Am. J. Ophth., **24**:322-323 (Mar.) 1941.

Schepens, C. L.: Retinal detachment and aphakia. Arch. Ophth., **45**:1-17 (Jan.) 1951.

Wadsworth, J. A. C.: Etiology and pathology. In symposium: Retinal detachment. Tr. Am. Acad. Ophth., **56**:370-397 (May-June) 1952.

NOTES, CASES, INSTRUMENTS

AN INSTRUMENT FOR HOLDING EXCISED FULL-THICKNESS DONOR CORNEA*

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Technically, it is difficult to cut a lamellar graft from an excised full-thickness donor cornea, especially when the graft is planned so that it will have the same diameter as that of the isolated cornea, since the tissue has to be fixed at its edge.

An instrument (fig. 1) has been devised to eliminate the difficulty. It consists of a circular metal platform with four equidistant arms radiating from its margin. A segment of metal tube is fixed to the proximal portion of each arm so as to form a tunnel to accommodate an ordinary small straight-toothed iris forceps. The roof of the tunnel is perforated by an adjustable screw, and the floor is serrated. Within the tunnel the forceps can be moved forward or backward in a horizontal plane, and its blades can be opened and closed in a vertical plane with the help of the adjustable screw. The platform can be mounted on a Tudor Thomas stand and locked in any position.

Fig. 1 (Basu and Ormsby). Instrument for holding excised full-thickness donor cornea

Procedure

The excised piece of the full-thickness donor cornea is placed on the platform with its endothelial side up and is secured at its periphery without traction by adjusting the forceps. The edge of the excised cornea, between two pairs of forceps, is grasped with a corneal forceps and a small horizontal incision is made with a knife, splitting the corneal stroma at the desired depth. A straight Tooke knife is then introduced through the incision and the cornea is split from the center to the periphery. By releasing anyone of the forceps and re-applying it whenever necessary, the margin of the graft can be split to the edge.

With the help of the instrument, smaller lamellar and full-thickness grafts may be trephined out from larger pieces of tissue on a thin flat piece of cork placed under the graft prior to securing its margin with the forceps.

The same instrument can also be used for cutting a graft from a piece of cornea with its epithelial surface upward, by placing a suitable plano-convex piece of cork between the endothelial surface and the platform, prior to fixing the corneal tissue with the forceps.

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A TELEVISION READER*

As a subnormal vision aid

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The majority of subnormal vision aids used for reading have as their basis the presentation of an enlarged clear image to the retina of the subject. This may be done in one of two general ways: (1) augment the optical system of the individual's eye by means of high plus lenses or telescopic lenses with reading caps, leaving the size of the print unchanged; or (2) utilize the patient's visual system with the usual correction and enlarge the print to be read. Devices in the second category ordinarily have been optical projectors which utilize either microfilm or the original printed material, as in the American Optical and the American Foundation for the Blind types.

The chief difficulty of such an optical projection system lies in the loss of contrast that necessarily goes with magnification. Further contrast losses are caused by light scattering within the ground-glass screen. If the screen is viewed at any angle other than the true, normal brightness losses result.

It occurred to us that the difficulty of poor contrast could be obviated by inserting electronic image intensification into the optical system. One possible way to do this is to view the reading material through a closed-circuit television system. Here magnification can be controlled by the optical system of the television camera and image intensification can be achieved electronically. The monitor screen acting as the effective light source has the additional advantage of having luminance relatively independent of the direction of observation by the viewer.

Such a device was constructed by using a Dage closed-circuit television camera equipped with a three-inch telephoto lens and a monitor which in this case was 14 inches in diagonal dimension (any standard television set may be used). The reading material was clipped to a movable stand which could be moved by a manual gear drive in any direction in a horizontal plane. Thus each line of print could be scanned in succession, using the mechanical drive. A photograph of this equipment is shown in Figure 1.

Magnification of 10 times with high contrast can be obtained with ease with the present equipment. However, there is no theoretic limit to the upper limits of magnification other than the size of the television monitor used.

One disadvantage of a television reader is the tendency for the image to persist due to the storage feature of the Vidicon camera tube used in this instrument. This is a minor inconvenience and by no means an insuperable one, if the instrument is sufficiently worthwhile on other grounds. Additional refinements, such as motor-driven transport for the reading material, foot switch control, fast return at the end of a line, and so forth, can all be incorporated with no modification of the essential components.

The Vidicon camera could also be replaced by a flying spot scanner. This would provide variable magnification without changing or moving the lens. The contrast would be unaffected. A single knob would thus permit the user to vary the magnification in accordance with his own needs and the type of material being scanned.

It might also be mentioned that the new transparent evaporated phosphors offer great promise for providing television picture tubes which may easily be viewed under normal

* From the Laboratory for Research in Ophthalmology and the Department of Anatomy, Western Reserve University, and the Ophthalmology Service, University Hospitals of Cleveland. This work was supported in part by a grant from the Diabetes Association of Greater Cleveland and the Cleveland Diabetic Fund. This apparatus was demonstrated before the East-Central Section of the Association for Research in Ophthalmology, January, 1958.

Fig. 1 (Potts, Volk, and West). A television reader.

ambient illumination with no appreciable loss in contrast.

One may anticipate that the present cost of about $1,000.00 for the components will be reduced as a result of the rapidly increasing demand for closed-circuit television systems. Some economy could be achieved by utilizing an existing television receiver as monitor. Although present costs would preclude the use of such a device for most individuals, public libraries or other institutions might well afford such an instrument.

University Hospitals (6).

A NEW INSTRUMENT FOR MUSCLE SURGERY*

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New York

This instrument† has been designed to combine the functions now carried out by three separate instruments. It looks like a muscle hook (fig. 1-A). At one end is the hook (fig. 1-C) which is composed of two parts, and at the other a knurled thumb screw (fig. 1-D). Turning this screw actuates a mechanism inside the handle, which opens the two halves of the hook end of the instrument (fig. 1-B). The distal half of the hook is smooth, and the proximal half has pins which fit into the holes in the distal half (fig. 1-E). When the two halves are brought together by turning the knurled piece with muscle or tendon between them, the instrument acts as

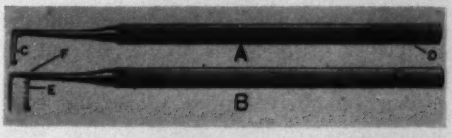

Fig. 1 (Castroviejo). Instrument combining the function of three instruments, hook A, C, clamp B, E, and caliper F.

* From the Department of Ophthalmology of St. Vincent's Hospital and the New York Eye and Ear Infirmary and New York University Post-Graduate Medical School.

† This instrument is manufactured by E. B. Meyrowitz, Inc., 520 Fifth Avenue, New York 18, New York.

a clamp. In addition, on the shaft of the instrument there is a scale in millimeters which makes it possible to use the instrument as a caliper (fig. 1-F). The calibrations measure the exact distance between the two halves of the hook when they are separated. By pressing the open ends of the hook against the sclera, distinguishable marks are made which indicate the points at which sutures are to be inserted.

9 East 91st Street (28).

CHARACTERISTICS OF ACCEPTANCE AND REJECTION*

Of optical aids in a low-vision population

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This study was undertaken under the auspices of The American Foundation for the Blind in order to ascertain the type of individual who would either reject or accept optical aids. It was our contention that differences in personality make-up were factors in success or lack of success with these aids. The results of this study seem to have certain implications for the clinical practice of ophthalmologists.†

A study of 60 visually handicapped persons indicated that a relationship does exist between personality characteristics and the acceptance or rejection of aids. A patient who was friendly, optimistic, active (a doer), neither submissive nor dominant but self-accepting, tended to accept optical aids. Whereas, a patient who was hostile, pessimistic (or unrealistically optimistic), inactive (a nondoer), submissive or dominant and self-rejecting, tended to reject optical aids.

The 60 patients (3 : 7) chosen were quoted for ophthalmologic disease, occupation, education, marital status, onset age, number of visits to a clinic, age, and sex distribution. The patients were chosen randomly from two populations of 500 patients each from previous investigations conducted at The Industrial Home for the Blind (1 : 18) and The Lighthouse (2 : 172), wherein acceptor and rejector groups had been established.

Methology

The psychologists sought to predict the personality patterns of each patient through the use of a focused interview, questionnaire, sentence completion test, and a thematic projection test. The psychologists predicted each patient's acceptor-rejector status without seeing the individual results of the agencies. Each patient was seen in the psychologists' offices for one visit. A follow-up interview established rejection and acceptance as postulated by the original studies conducted at The Industrial Home for the Blind and The Lighthouse.

Discussion

This study revealed that basically three types of individuals are involved in the acceptance or rejection of optical aids: (1) the "acceptor," (2) the "mixed type" who is not clearly accepting or rejecting in his orientation to life, and (3) the "rejector."

The patient who is an acceptor type is one who is able to come to grips with life and its varied problems. He is able to perceive situations rationally with a minimum of unnecessary neurotic involvement. Such a person is friendly, generally accepting people, amenable to suggestions, tends to perform many tasks on his own, and has a realistic appraisal of his condition and aspirations for the future. Such a person is capable of utilizing an aid with an understanding of its limitations and how it can be most suitably applied in specific occupational, educational,

* This study was aided by a grant from The American Foundation for the Blind.
† We are indebted to Dr. Gerald Fonda for his helpful advice and suggestions concerning low-vision patients.

or social pursuits. The acceptor can readily be prescribed for in an ophthalmologic setting, and will more than likely cause the least amount of time investment both in terms of re-education and clinical personnel. In sum, he comes to the office or clinic, wants to be helped, and will attempt to adjust whenever feasible.

The mixed type is not clearly an acceptor or rejector. He may be confused as to what the aids will accomplish, and he may be unsure of his reasons for coming to the ophthalmologist. Such an individual, for example, may be pessimistic about his future, unfriendly, not accepting of his handicap, yet clearly demonstrate a doing and active quality to his background. It is not unusual to find such a person to be among a group of recently blinded, and one who has not fully learned to adjust to his present condition. The mixed type will need to be handled cautiously, may require numerous educative visits to the office, and may necessitate the introduction of other clinic personnel, for example, social worker, counselor, psychologist, depending upon the area in which this patient's problems seem focused.

With the rejector it is not only necessary to move slowly, but it may be necessary to establish a relationship over a long period of time before such a person is assisted. This type of patient tends to be negativistic, hostile, and may be quite pessimistic. He tends to be overbearing in his attitude at one time and seemingly submissive and compliant on another occasion. He is rejecting of himself to the point of self-depreciation and more than likely unaccepting of his eye condition. The rejector usually enters the clinic in a negative mood, may believe that if he improves he will lose a pension, agency allotment, or other financial support that he may be receiving. This individual seems to need his disability possibly in order to control others, receive psychologic reassurance, and uses the eye condition to act out his dependency needs. The ophthalmologist ought to look upon the overt reactions of this type of patient as being a cover for deeper emotional disturbances and anxieties which may necessitate counseling, guidance, or psychotherapy. In sum, the rejector will require the greatest amount of energy, time, and clinic personnel involvement. He needs to be shown that he can be helped, and that the acceptance or trial of an aid will not be threatening to him.

Further conclusions drawn from this study suggest that the criteria and definition of success in terms of optical aid prescription need to be clarified. Success as defined by previous investigators was evaluated in terms of the acceptance or rejection of an aid. Follow-up investigation in this study, through the use of a telephone interview, indicated that over a period of time patients who had been considered rejectors became acceptors. This description is purely from a psychologic point-of-view and we are assuming an adequate diagnosis and a technical efficiency of the prescriptions.

Two important factors seem to account for this change. One, the goal of the ophthalmologist may have been set too high for the patient, and, secondly, the level of aspiration of the patient himself may determine the degree of success. If, for instance, a patient desires a magnifier for work only and he cannot be assisted, then such a person might be considered a rejector, whereas a few months later this same patient may come in and accept a reading aid, and be then classified as an acceptor.

In other words, level of success in optical aids prescriptions ought to be viewed in terms of adequacy of the ophthalmologist's goals and the needs and aspirations of the patient. We defined success as any movement on the part of the patient from one aid to another, or from no aid to any aid, regardless of ophthalmologic opinion on how a patient could best be assisted.

A further consideration of this study was to suggest the possible types of questions or statements that could be utilized by an ophthalmologist in daily practice in order to elicit information that would enable him to

classify a patient as either an acceptor or rejector. For example, the open-end question might be utilized in such a way as to elicit feelings, opinions, and personality orientations.

Questions such as: *How* do you *feel* about your _____?, *What* is your *opinion* on _____?, *How* do you get along with _____?, *What* causes difficulty in _____?, and *What* do you think about _____? are helpful in ascertaining personality make-up. It is important to point out that a direct question or statement calling for a "yes" or "no" response may lead to little significant information and possible evasiveness on the part of the patient.

Summary and Conclusions

This article has dealt with the types of personality structure which tend to accept or reject optical aids. It was determined that the friendly, optimistic, active, neither submissive nor dominant, and self-accepting person tends to accept optical aids, whereas the hostile, pessimistic (or unrealistically optimistic), inactive, submissive or dominant and self-rejecting individual tends to reject optical aids.

A re-evaluation of degrees of success was presented. The relation of the level of aspiration on the part of the ophthalmologist and patient to the criteria of success was clarified.

Practical considerations in identifying these personality types and a discussion of possible questions that the ophthalmologist might utilize in further eliciting personality make-up were presented.

890 Park Avenue (21).

References

1. Esbin, L.: A preliminary report of the first 500 clients served. The Optical Aids Service of The Industrial Home for the Blind, April, 1957, p. 8.
2. Fonda, G.: Report of 500 patients examined for low vision. Arch. Ophth., **56**:171-175, 1956.
3. Freudenberger, H. J., and Robbins, I.: The relationship between five selected personality characteristics on the acceptance or rejection of optical aids in a low vision population. Monograph to be published by The American Foundation for the Blind, 1959.

Ophthalmic Miniature

Public opinion, which on medical subjects is generally erroneous, although for the most part founded on professional authority, is in no instance more injurious than in relation to the eye. It pronounces it to be an organ of very delicate nature, equisitely sensible, requiring the greatest delicacy of touch, and the utmost nicety of management; which opinion some oculists formerly found it convenient to support, and which the public may still continue to believe without any great disadvantage; but students in surgery must be taught otherwise.

G. J. Guthrie, on "*The Certainty and Safety* with which *The Operation* for the *Extraction of A Cataract* from *The Human Eye* may be performed and the means by which it is to be Accomplished."

W. Sams, Royal Library, St. James Sheet, 1834, pg. 10.

OPHTHALMIC RESEARCH

EDITED BY FRANK W. NEWELL, M.D.

Abstracts of papers presented at the meeting of the East-Central Section of the Association for Research in Ophthalmology, Inc., at the Netherland Hilton Hotel, Cincinnati, Ohio, January 5, 1959.

Vergence and accommodation: III. Proposed definitions of the AC/A ratios. Mathew Alpern, Ph.D., Wilfred M. Kincaid, Ph.D., and Marvin J. Lubeck, M.D., Departments of Ophthalmology and Mathematics, University of Michigan, Ann Arbor.

Previous studies have investigated the convergence associated with a change in the accommodation response. The present paper will propose a method of quantification of the relation between these two responses. Use is made of an intervening variable the AC/A ratio. Two such quantities are proposed: (1) the amount of vergence associated with a unit change in the accommodation stimulus (the stimulus AC/A) and (2) the amount of vergence associated with a unit change in accommodation response (the response AC/A).

The basic assumptions are that motor innervation to the ciliary muscle and to vergence movements have a common point of origin, and that there is a linear relation between the magnitudes of these two innervations. Experimental determinations of the amount of vergence associated with a given stimulus to accommodation and the amount of accommodation so associated will be reported for four observers over the entire gamut of conceivable accommodation stimuli.

The data are in good agreement with the basic assumptions. Curves which relate vergence to the stimulus, accommodation to the stimulus, and vergence to accommodation may be drawn and when the gamut of stimuli are sufficiently large none of these curves are linear. It is possible to explain the forms of these curves, however, within the framework of the basic assumptions, in terms of known physiologic phenomena. By judicious selection of the range of accommodation stimuli all of the curves are linear. Such findings can probably account for the occasional case of nonlinearity reported in the literature.

The least square solution of the linear part of the curve relating the accommodation to the stimulus is obtained, as is least square solution of the linear part of the curve relating vergence to the stimulus. The slope of this latter curve represents the stimulus AC/A. Simultaneous solution of these two equations permits the identification of a third linear equation which relates vergence and accommodation and the response AC/A is one constant in this latter equation. The relation between the two types of AC/A ratios will be described. Clinical implications of these findings will be discussed.

The effect of sympathomimetic drugs upon the amplitude of accommodation. Robert D. Biggs, M.D., Mathew Alpern, Ph.D., and Donald R. Bennett, M.D., Departments of Ophthalmology and Pharmacology, University of Michigan, Ann Arbor.

Recent experiments have emphasized that the sympathetic nervous system has some role in ocular accommodation. Measurements were made (using the method of stigmatoscopy) of the accommodation response to a variety of accommodation stimuli following topical application of phenylephrine hydrochloride (Neosynephrine), cylopentolate hydrochloride (Cyclogel), as well as following subconjunctival injection of epinephrine bitartrate, and (in one case) epinephrine bitartrate with xylocaine hydrochloride (Lidocaine).

Subconjunctival epinephrine produced a loss of accommodative ability. The effect upon the near-point was much more marked than the (slight) effect on the far-point. When the injection of epinephrine was combined with Xylocaine in a single case, the loss of accommodation was almost complete, but again, the change at the far-point was slight. Time studies show that these changes were transient, the entire process was usually back to normal within 90 minutes.

The time course of the sympathomimetic drug effects were studied by a subjective Badal optometer in which the near-point of accommodation was measured under conditions in which such factors as pupil size, target size, and luminance are controlled.

The results can be interpreted either as a direct action on ciliary muscle innervated by the sympathetic nervous system, or conversely as a change in volume of the ciliary body under the influence of drugs which effect its vascular supply. The relative importance of these two alternatives will be discussed.

A mechanism for the sympathetic control of visual accommodation. David G. Fleming, Ph.D., University of Kansas, Lawrence, Kansas. (Present address, General Electric Company, Lamp Development Department, Nela Park, Cleveland 12, Ohio.)

An investigation was undertaken to test the hypothesis that the sympathetic portion of the autonomic nervous system may influence accommodation by regulating the tone of the blood vessels in the ciliary body. Since the ciliary body is a highly vascularized structure, changes in its blood

volume would alter its size and ultimately the tension exerted by the zonule on the lens. This is a mechanism supplementary to the direct stimulation of the ciliary muscle by autonomic effectors.

The experimental procedures followed two courses:

1. In a group of rabbits changes in blood flow through the ear following superior cervical ganglionectomy were compared with changes in accommodation. Ear temperature was used as the index for blood flow and skiascopy for refractive power. A time correlation existed between changes in ear temperature and refractive error. Preoperative ear temperatures were identical. On the first postoperative day the ears on the operated side were 2.5°C. warmer than the ears on the contralateral side. Within three days the difference disappeared. On the first postoperative day the eyes on the side with the lesion were 1.25 diopters less hyperopic than the eyes on the intact side. Within three days this difference also disappeared. One month postoperatively the rabbits were given 0.5 mg. Priscoline daily intravenously. Prior to the Priscoline test period ear temperatures and refractive errors were the same bilaterally. An average difference of 1.9°C. between the ears and 0.82 diopters between the eyes resulted, with the ears on the operated side always the warmer and the eyes always the less hyperopic.

2. A method was developed for studying gross and microscopic changes in the eye during stimulation of the cervical sympathetic nerve. The Richins-Hall toluidine-blue method for neural activity was adopted to this investigation. In the first series of cats, the cervical sympathetic nerve on one side was stimulated continuously for three hours. Just before the stimulation was discontinued, the anterior chamber of both eyes was perfused with lead subacetate. This produced instantaneous in vivo fixation of the eye. Gross examination of the eyes in midsagittal section revealed that the lens in the eye on the stimulated side was flatter than the lens in the contralateral eye. Histologic examination of the ciliary region demonstrated constricted vessels only in the stimulated eye. Both eyes however, indicated considerable metabolic activity. In a second series, the ciliary ganglion was extirpated unilaterally. The pupil in the eye on the ganglionectomized side was extremely dilated, indicating unopposed sympathetic activity. After three hours' stmulation of the contralateral sympathetic nerve, vasoconstriction was demonstrated only in the stimulated eye. As contrasted to the preceding series, a high level of metabolic activity was seen only in the stimulated eye. The results support the hypotheses that one means by which the sympathetic nervous system affects accommodation is through the vasomotor mechanism. In addition, unilateral cervical sympathetic stimulation appears to produce considerable parasympathetic activity in both eyes.

The elicitation of fusion in strabismus patients who are aware of diplopia. Samuel C. McLaughlin, M.S., Department of Ophthalmology, University of Michigan Medical School, Ann Arbor.

Strabismus patients who are aware of diplopia but who cannot fuse are of particular interest for the theory of normal and strabismic binocular vision because of the apparent absence of any obstacle to fusion. In this paper, a method for treating such patients successfully will be described in detail. The method consists of the following steps:

1. The patient is taught to alternate fixation while aware of diplopia, thus causing each image in turn to be seen "straight ahead," so that the two images appear to move back and forth. Evidence will be presented that this visual exercise causes the two images to be seen as progressively more similar.

2. Using polarized lights in a dark room, with each light seen from one eye, the patient is taught to perceive the two in the same place at the same time when the distance between them subtends the angle of strabismus. This is facilitated by having one of the two lights in the form of an annulus and the other in the form of a small dot of contrasting color.

3. This perception is gradually transferred to a bright room and then to objects. The patient is thus taught to perceive two objects in the same place at the same time when the objects are separated in space by the angle of strabismus. Later, identical objects (for example, two coffee cups from the same set) are viewed in this way, and the patient is taught to fuse them—to perceive the two as one.

4. The patient is now able to fuse with the aid of an ophthalmic prism. He is given extensive practice in maintaining such fusion for longer and longer periods of time.

5. The patient is now taught to fuse without the prism by means of the following technique (assume the patient to be an esotrope of 10 diopters):

a. A prism of approximately 15 diopters is placed base-in before one eye, and the other eye is occluded (clip-on occlusion is sufficient). The patient wears this prism-occluder combination as much as possible, thus forcing adduction of the dis-occluded eye.

b. Each time the prism-occluder combination is removed, the patient again practices fusion with the aid of a base-out prism. It is invariably found that, over a period of several weeks, progressively less base-out prism is needed for this purpose, and the patient is soon able to fuse at any distance without the aid of any base-out prism.

6. The patient is then given extensive practice in maintaining fusion without prisms for longer and longer periods. At the same time, the patient is given increased prism-vergence amplitude by an extension of the technique described in paragraph (5).

On the relationship between corneal hydration and transparency. Albert M. Potts, M.D., and

Beatrice Cohen Friedman, B.S., Laboratory for Research in Ophthalmology, Western Reserve University, Cleveland 6, Ohio.

Studies were done on fresh beef corneas excised with a ring of sclera and clamped by means of the scleral ring into a special cell. This cell allows complete control of the environmental factors of temperature, pressure, tonicity, and chemical composition at each limiting membrane. At appropriate times measurements were made of weight, thickness and transparency; and water content was determined at the end of the experiment.

Marked differences in maintenance of transparency were found between corneas bathed in solutions of the same composition on each side and those where epithelium is hypertonic to endothelium. Perhaps most striking of all is the large contribution of epithelium to early clouding, particularly that occurring after denuding of endothelium. The findings require some adjustment in our thinking about the direct relationship between corneal hydration and transparency.

The influence of tonography of one eye on the tonographic readings of the fellow eye. Amerigo Cambiaggi, M.D., and W. M. Spurgeon, Ph.D., University of Cincinnati Medical School, Cincinnati, Ohio.

The studies were carried out on 32 normal eyes (16 subjects and 48 eyes affected by chronic simple glaucoma (24 subjects), using a Mueller electronic tonometer. On the first day tonography was performed first on the right eye, then on the left eye. After two or three days, using the same subjects at the same hours of the day, tonography was performed first on the left eye, then on the right eye. Two series of experiments were performed. In Series 1 (12 normal, 14 glaucomatous eyes) the time interval between tonographies was two minutes. In Series 2 (20 normal, 34 glaucomatous eyes) the time between tonographies was four minutes. The resulting data were analyzed statistically.

The data indicate, but with these small samples do not prove, that in a group of individuals the average intraocular pressure for either right or left eyes, normal or glaucomatous, is decreased by prior tonography on the contralateral eyes. The decreases ranged up to 7.0 mm. for normal eyes and 8.8 mm. for glaucomatous eyes, but increases up to 4.2 mm. for normal eyes, and 4.3 mm. for glaucomatous eyes were also observed.

The average facilities are not significantly changed in either right or left eyes by prior tonography on the contralateral eyes. This is true for both normal and glaucomatous eyes.

The average flow in normal left eyes is significantly higher (five-percent level) when tonography is performed on these eyes first, with four-minute intervals. The trend is in the same direction for normal right eyes with the same intervals but the difference is not significant. None of the differences between average flow is significant for normal eyes at two-minute intervals, or for glaucomatous eyes at either interval.

In individual subjects both increases and decreases are noted for all tonographic values following tonography on the contralateral eye. Therefore no general rule can be given to cover all cases.

Comparative secretory nature of ciliary process and choroid plexus tissues. Morton B. Waitzman, Ph.D., and Elmer J. Ballintine, Department of Pharmacology, School of Medicine, Western Reserve University, Cleveland, Ohio.

Certain morphologic and functional comparisons between ciliary process and choroid plexus tissue will be discussed. Although there is wide acceptance of the role of these tissues in aqueous humor and cerebrospinal fluid production, final proof for this role, particularly in the case of choroid plexus, is yet lacking.

Some of the earliest work of Friedenwald and others reported on metabolic structures common to both these tissues, such as the cytochrome and succinoxidase systems. In our laboratory a specific type of adenylic acid deaminase system has been elucidated for both ciliary process and choroid plexus. A study of the ion distribution in aqueous and cerebrospinal fluids compared with a plasma filtrate would indicate certain similarities concerning an actively transported sodium, but the distribution of other ions, such as chloride and potassium, complex the metabolic interpretations. Clearly, the metabolic roles played by ciliary process and choroid plexus tissue are similar, but not identical.

A contribution to the injection technique for studying retinal blood vessels. H. R. Hausler, M.D., and T. M. Sibay, M.D., Departments of Ophthalmology and Physiology, University of Toronto, Toronto, Canada.

By injecting the retinal vessels with silver-nitrate solution and subsequent exposure of the eye to a bright light, portions of the vessel walls, particularly the intracellular cement substances and reticular fibers, are stained brown. Thus the retinal vascular tree can be studied in its continuity and also structural details observed in the vessel walls.

Studies on the visual toxicity of methanol: X. Further observations on the ethanol therapy of acute methanol poisoning in monkeys. Anita P. Gilger, M.D., I. S. Farkas, M.S., and A. M. Potts, M.D., Laboratory for Research in Ophthalmology, Western Reserve University Cleveland 6, Ohio.

In previous work we showed that repeated, small oral doses of ethanol started simultaneously with a single LD_{50} of methanol prevented both death and severe acidosis in monkeys. The present papers reports:

1. Results of delayed ethanol therapy of acute methanol poisoning in monkeys.
2. The effect of delayed ethanol therapy on acidosis and blood methanol levels.
3. An estimate of how much delay may be tolerated in treatment of primate methanol poisoning with ethanol.

Field trial of the Hardy-Rand-Rittler and other color vision tests. Clement McCulloch, M.D., and N. C. Turnour, Toronto, Canada.

A field study was made to compare the Hardy-Rand-Rittler Color Vision Plate Test with the American Optical 15 Plate Edition Pseudo-Isochromatic Plate Test and the RCAF lantern.

Of a total 3,508 subjects tested, based on a revised or most probable classification, 16.5 percent of the population tested, were found to have color-defective vision by H-R-H testing and 5.6 percent by the other test method. It is clearly indicated that reliable color-vision test results depend not only upon the test used but also on the method of administering the test. Reliable results should be obtained, providing the H-R-R test is properly administered. A suggested revised method of recording responses is used.

Photographing the eye by means of slitlamp illumination. S. J. Vaile, M.D., W. P. Callahan, M.D., and A. Smialowski, Toronto, Canada. Department of Ophthalmology and the Medical Photography Department, St. Michael's Hospital and the University of Toronto.

The Haag-Streit slitlamp has been altered to enable us to take 35-mm. color transparencies of many eye conditions.

A 300-watt second electronic flash is incorporated in the illuminating system. The binocular microscope is replaced by a Leica camera with a 135-mm. lens in a reflex housing. The film used is Super-Anscochrome. Color transparencies illustrating the advantages of oblique and slitlamp illuminations will be shown.

Conoid cataract lenses for the correction of aphakia. David Volk, M.D., Western Reserve University, Cleveland 6, Ohio.

Aphakic patients wearing strong spherical lenses have two main complaints: marked blurring of vision when looking through peripheral parts of the lens, and marked distortion. Distortion is manifest in two ways: (1) when the eye is stationary and looking straight ahead and (2) when the head and eyes are turning for fixation. (1) and (2) are similar but the effects in (2) are severely exaggerated.

In a previous paper the use of conoid surfaces was discussed with reference to producing lenses which have a minimum of aberrations. The same principles have been employed to produce a series of cataract bifocal lenses which give an optimum correction of the annoying distortions and blurring of spherical cataract lenses. The primary consideration was the production of clear vision throughout the entire lens. This requires that lateral overcorrection and marginal astigmatism be eliminated. The use of elipsoids of revolution as the front surfaces of the conoid cataract lenses makes it possible to produce lenses in which the effective power of the lens for the rotating eye behind the lens is the same for all positions of the eye. The correction of the aberrations includes the reading segment area so that reading vision is extremely clear, enabling an increased reading range.

Distortion is reduced to the point where it is negligible. As a result of the nearly uniform magnification produced by conoid cataract lenses, there is an increase in the size of the field of vision as compared to that of spherical cataract lenses of the same diameter which have nonuniform magnification.

Clinical evaluation and subjective patient response to the wearing of conoid cataract lenses indicate a remarkable improvement in the quality of the vision over that with spherical cataract lenses.

Acuity measurements throughout the various zones of conoid cataract lenses will be shown graphically for several patients and these results compared to similar measurements on the same patients wearing spherical cataract lenses.

Conoid lenses for indirect ophthalmoscopy. David Volk, M.D., Western Reserve University, Cleveland 6, Ohio.

Conoid lenses have been specifically designed for use as the condenser-image forming lenses in indirect ophthalmoscopy. These conoid lenses differ from the conoid lenses used in subnormal vision in that the aspheric surface necessary for ideal image formation in indirect ophthalmoscopy is an elipsoid of revolution of higher degree of eccentricity. This is necessary because of the increased distance between the eye and the lens.

Th use of conoid lenses for indirect ophthalmoscopy has two important results:

1. As a condenser lens. Light from the ophthalmoscope is refracted more efficiently towards the entrance pupil of the eye. Consequently, a greater proportion of the light will pass through the pupil to illuminate the fundus.

2. As an image former. The aerial image of the fundus is extremely clear and the entire lens is useful in image formation.

Whereas the spherical condenser-image-forming lenses cannot be used in large diameters or high powers because of the poor quality of image formation, the conoid lenses can be used in higher powers and in relatively large diameters, producing a larger and clearer aerial image for a particular power.

The ease with which a clear aerial image can be obtained with conoid lenses makes the entire procedure of indirect ophthalmoscopy considerably less difficult than it is with spherical lenses.

A short review of the optics of indirect ophthalmoscopy will be given, explaining image size, magnification, and position of the aerial image in ammetropia. In particular, the optics of the conoid lens as the image former will be discussed.

Oxidized pyridine nucleotides in corneal epithelium. Nina H. Morley, Ph.D., Department of Ophthalmology, University of Toronto, Toronto, Canada.

The estimation of oxidized pyridine nucleotides (oxidized coenzyme I and II) has been studied in beef and rabbit corneas by scraping epithelium from the eyes frozen in a mixture of petroleum ether and dry ice or in liquid nitrogen, extracting it in trichloroacetic acid and analyzing the extract fluorometrically. The concentrations per gm. of wet tissue, as DPN, average 1030 μg. for beef and 650 μg. for rabbit corneal epithelium.

These values are higher than any reported elsewhere for other tissues. Concentrations are also to be expressed in terms of dry weight and protein content of the tissue.

Pathology of cornea guttata. J. Reimer Wolter, M.D., and Bertil F. Larson, M.D., University of Michigan, Ann Arbor.

Earlier studies have revealed that hyaline excrescences which are found on the posterior surface of Descemet's membrane in cases of Fuchs' endothelial and epithelial dystrophy are the histologic equivalent of the guttae seen with the slitlamp. These hyaline excrescences are the product of a primary degeneration of the corneal endothelium.

A peculiar kind of cornea guttata was observed following different types of deep keratitis. The hyaline excrescences in these cases formed lines and bizarre geometric patterns. In one case it was possible to study the histopathology of these changes for which the term "secondary cornea guttata" is suggested.

Ophthalmic Miniature

Treatment for Night Blindness: Bleeding at the forearm; purgation of the belly by means of medicine and clyster. Then the head must be cleared by gargling and sneezing and the veins in the inner corners of the eye must be bled, and he (the patient) must drink before a meal (water with) dry hyssop or rue. It (the eye) must be anointed with alum, rock salt and the juice which flows from the goat's liver when it is roasted, and he must admit to the eyes the steam rising from it during the roasting and then eat (the liver).

"Treatment of Night Blindness,"
from *The Ten Treatises on the Eye,*
by Hunain Ibn Is-Haq, 809-877, A.D.

SOCIETY PROCEEDINGS

Edited by DONALD J. LYLE, M.D.

OXFORD OPHTHALMOLOGICAL CONGRESS

July 7-9, 1958

SIR J. W. TUDOR-THOMAS, *Master*

DOYNE LECTURE

The annual Doyne Lecture, in memory of the founder, was delivered by O. GAYER MORGAN, F.R.C.S., ophthalmic surgeon to Guy's Hospital, London, on the subject of "The early clinical diagnosis of chronic glaucoma." Reference was made to the high incidence of blindness from this disease and to its increasing incidence in the population over 40 years of age. With routine tonometry, Read and Bendor-Samuel and others report that as many cases are undiscovered as are diagnosed and treated in this age group. The problem of how to reach this mass of potential disease is one in which the general practitioner could, if sufficiently trained in awareness, be of very great value. It is significant that 75 percent of this group of patients at present have their sight tested first by sight-seeing opticians, who are not instructed in the recognition of the abnormal. The lecturer considered the many aspects of the problem on a national scale.

VASCULAR CHANGES AND VISUAL FAILURE

The first discussion was on "The influence of vascular changes in progressive failure of vision," the opening papers being contributed by SIR G. W. PICKERING (Oxford), DR. R. LEISHMAN (Glasgow), and MR. L. H. SAVIN (London).

Sir George's paper was read by Dr. Pears in his absence owing to illness, and drew attention to the importance of occlusion of the internal carotid in many patients in this group in whom sudden loss of vision was a presenting feature.

Dr. Leishman's thesis was to challenge the concept of spasm, especially in respect of the retinal vessels; he could not convince himself that it occurred. Instead, he drew attention to changes of fibrotic replacement, to the development of atherosclerotic patches, and to the frequency of distant factors in failure of peripheral circulation. General vasodilator treatment might prove a further embarrassment to the retinal circulation when small vessel walls were fibrotic and the blood column already meager.

Mr. Savin dealt with the problem from the view of the clinical ophthalmologist, and reviewed and differentiated a number of the vascular conditions causing progressive visual loss, with particular attention to intracranial defects and general diseases of the peripheral blood vessels.

A lively general discussion followed, to which the openers finally replied.

TOPICAL ANTIBIOTICS

MR. FREDERICK RIDLEY (London) read a paper on "The rational use of topical antibiotics in ophthalmology." Careful investigations were undertaken to estimate the actual concentration, persistence, and penetration of a number of drugs under conditions of outpatient or home treatment. The relationship to tissue sensitivity and the question of the production of resistant strains were also dealt with. The conclusion arrived at was that penicillin in high concentration, used for short periods, was infinitely more effective than other antibiotics tested, and that sensitivity and resistant strains were not experienced in these circumstances.

RETINAL DETACHMENT

MR. P. A. GRAHAM (Manchester) discussed two cases of retinal detachment showing very low tension, aqueous flare, posterior synechias and concentric folds in the iris. The

detachments were annular and seen in the pars plana.

Larger corneal grafts

Mr. Derek Ainslie (London) read a paper advocating the use of the larger corneal graft in many cases and showed a number of good results of seven mm. and more diameter.

Skin grafting to orbit

Mr. L. R. McLaren (Manchester) described a technique for immediate skin grafting to the entire orbital cavity in cases of exenteration for malignancy.

Electroretinography

Professor Karpe (Stockholm) discussed electroretinography and indicated the clinical conditions in which it might be of value diagnostically or in the assessment of a prognosis. The technique could be of value in cases of retinal detachment, occlusion of the retinal vessels, in hereditary tapetoretinal degeneration, and in retinitis pigmentosa: also in inflammatory diseases and in siderosis bulbae.

Development of lens

Dr. D. Stenhouse Stewart (Hull) presented on behalf of Prof. Chanterishvili Tiflis (U.S.S.R.) a treatise on the "Development of the crystalline lens."

Instruments

Mr. Lloyd Johnstone (Worcester) showed a simple, new, mobile operating lamp.

Mr. D. P. Choyce (Southend) described and illustrated the use of Strampelli-type anterior chamber implants and gave his results.

Dr. H. J. Flieringa (Rotterdam) described an ingenious metal scleral ring for supporting the eyeball at operation and thus preventing loss of vitreous.

Socket contraction

Mr. G. J. Romanes (East Grinstead) gave a paper on the "Treatment of socket contraction," including its pathology and complications and an analysis of 60 cases. The principles of operative techniques were fully shown.

Keratoplasty

Dr. Townley Paton (New York) gave a masterly survey of some "Complications and pitfalls in keratoplasty," full of valuable practical comment and advice, covering instrumentation, the state of the recipient and donor eyes, and the preoperative and postoperative course.

The last item on the second afternoon was a session of "Any questions"; members submitted questions and the audience spontaneously provided the answers, with comment and discussion.

Uveitis

The chief item of the final morning was a discussion on "The etiology and treatment of uveitis," opened by Mr. E. A. Perkins (London), Mr. A. Sanworth (Manchester), and Dr. R. D. Catterall (London). The former gave an account of work done at the Uveitis Clinic of the Institute of Ophthalmology, emphasizing the probability of toxoplasmosis in a high proportion of cases of posterior uveitis; while Dr. Catterall drew attention to the importance of prostatic investigations in males showing anterior uveitis, a number of which might be Reiter's disease. Mr. Stanworth dealt with the clinical picture and showed the changing incidence of etiology between granulomatous and nongranulomatous infections.

Chronic simple glaucoma

This discussion was followed by two papers on chronic simple glaucoma. Dr. Leydhecker (Bonn) examined critically the basis of tonography and advanced reasons for some modifications which would give greater reliability.

Dr. S. M. Drance (Saskatoon) dealt with the water-drinking provocative test as a means of hemodilution. Depending on the in-

itial blood-picture of any individual patient, which must first be determined in each case, this test could be of value in early cases of simple glaucoma.

NEW ENGLAND OPHTHALMOLOGICAL SOCIETY

442nd meeting, March 19, 1958

DR. VIRGIL G. CASTEN, *Presiding*

INFLUENCE OF SYSTEMIC STEROIDS ON INTRAOCULAR PRESSURE

DR. PEI-FEI LEE, Boston: A group of 13 severe asthmatic patients with no history of ocular disease were given ocular examinations which included tonography and gonioscopy while on long-term systemic steroid therapy. Only one of these 13 patients showed a sign of early open-angle glaucoma. What was seen in the other patients could well represent the incidental findings found among people over 40 years of age. No trend toward glaucoma was found in the group as a whole. The average values of intraocular pressure was 20.35 mm. Hg; facility of outflow was 0.24 cu. mm./min./mm. Hg; rate of aqueous flow was 1.63 cu. mm./min. This compares well with the findings in the normal population. There was nothing to indicate that systemic steroid therapy had a significant tendency to elevate the intraocular pressure or impair the facility of outflow.

USE OF AVERTIN ANESTHESIA IN INTRAOCULAR SURGERY

DR. JOHN M. MCIVER, Quincy: Avertin as an anesthesia offers several advantages: (1) there is a marked hypotony, probably due to lower intracranial pressure and lower pressure and dilatation of the small blood vessels and capillaries of the orbital and ocular bed (2) Avertin may be given to the patient in his room; (3) postoperative nausea is either none or minimal; (4) there is no great risk of postoperative psychosis; (5) the possibility of retrobulbar hemorrhage is eliminated because retrobulbar injection may be dispensed with, four percent cocaine topically seems to be adequate; (6) there are no pulmonary irritations, coughing and sneezing are minimized.

In the last eight years I have employed Avertin for intraocular surgery on 24 eyes and assisted others when it was used in at least four more eyes. In these cases there were no deaths, no pneumonia, no embolic strokes, no hemorrhages, no infections, no delay in the reformation of the anterior chamber, and no loss of vitreous.

In my cases the most frequent indication for Avertin was apprehension and anxiety on the patients' part. It was also indicated for those patients whose physical habitus suggested the likelihood of vitreous prolapse. It was also used for those patients with high myopia and known fluid vitreous.

I would like to say that in cases of intraocular surgery where local anesthetics do not seem to be the methods of choice, I would recommend Avertin.

BETA RADIATION: INDICATIONS AND COMPLICATIONS

DR. BRENDAN D. LEAHEY, Lowell: It should be borne in mind that beta radiation is a two-edged sword. Its use entails the possibility of doing very grave damage to the ocular tissues. The overzealous overtreatment of earlier years should not be repeated. Radiation should not be used on any eye where a similar effect can be obtained by other means of treatment.

Despite all the complications in our cases it should be pointed out that nearly all the eyes treated were originally blind, deeply scarred, or heavily vascularized. Since their only hope was this treatment, relatively little harm was actually done. In general, the results in this series of 320 cases were remarkably good.

Beta radiation when administered properly is a very valuable adjunct to ophthalmic treatment and constitutes one of the greatest advances in ocular therapy in recent years.

RETINAL VENOUS OBSTRUCTIVE SYNDROME

DR. GEORGE N. WISE, New York: This theoretical concept has a good deal of clinical and some pathologic and histologic material to substantiate it, It has certainly given me a better understanding of a number of vascular diseases that previously had been confusing to me.

The background of this concept can be found in Michaelson's beautiful work on the embryology of the retinal circulation. From his data Michaelson hypothesized that the blood vessels grew in response to some factor—some metabolic factor—we will call the vasoproliferative factor. This factor was present only in areas where retinal anoxia existed and the factor was in the form of a gradient so that the vascular channels grew in an orderly fashion. When the vascular meshwork reached the ora and the retina was completely oxygenated the vasoformative factor disappeared and the vascular development ceased.

Michaelson speaks of a state of equilibrium existing and he also states that this state of equilibrium can be disturbed in the adult retina. This disturbance is brought about under certain pathologic conditions and thus new veins, vessels, and capillaries can be formed.

Our theoretical concept is that the basic lesion underlying the upset of this delicate balance is some degree of capillary or vein obstruction. Like Michaelson we tend to consider the capillaries and veins as one unit in regard to this factor of upsetting the equilibrium. If there is a group of obstructed capillaries there may be a local area of retinal hypoxia. The vasoformative factor will reform there and there will be vasoproliferation in one little locus. If it is more widespread, or a larger vein is involved, then the area drained by that vein will become stagnant. A relative retinal anoxia will develop in the area, the vasoformative factor will reappear, there will be neovascular formation, and a fibrous supporting tissue will grow out. Regional capillaries and veins of the area will try to re-establish circulation.

In almost all the diseases we studied that showed neovascularization we could see microaneurysms appear. The one exception was retrolental fibroplasia. This gave us a new concept for microaneurysms: these microaneurysms were budded or aborted neovascular formations.

Charles Snyder,
Recorder.

OPHTHALMIC MINIATURE

Would you believe that, at the age of 15 or 16, when I read a book on the habits of birds, I was deeply anxious to know how the author was able to discuss all the details? "After all," I told myself, "one doesn't see those birds, one hears them sing, and so one knows that they exist. But nobody has ever seen a bird in a tree."

Chapter on myopia in *Gare à vos yeux*
by Francisque Sarcey, Paris, 1884.

AMERICAN JOURNAL OF OPHTHALMOLOGY

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AUDIENCE APPEAL

The following suggestions for the presentation of scientific papers were prepared by Frederick C. Cordes for use of the overworked secretary of the American Ophthalmological Society. Each essayist will receive a copy of these suggestions as "hints" for the benefit of his listeners.

As is usual of Dr. Cordes, these remarks are full of common sense and represent many years as a teacher and essayist. I feel certain that our readers who will follow these suggestions will receive the gratitude of their audience and will make friends and influence people. (D. T. V.)

SUGGESTIONS FOR PRESENTATION OF PAPERS

The average medical paper is too long to be presented in toto in the time allotted for this purpose. If the highlights are to be brought out adequately, it is necessary that a second paper be written that will come within the allotted time. Don't thumb through your detailed papers or just read parts. The effect is always a disjointed paper and makes your audience feel you haven't bothered to prepare the presentation properly.

Read the paper you intend to present out loud several times to four walls so that you know the time it takes and so that you are familiar with it. This includes the slides used, as it is important to be familiar enough with these that it won't be necessary to stop to refer to a slide list. The paper, as "read to four walls," should be two minutes shorter than the allotted time.

Slides are often an important part of a paper. Use only good slides. Be familiar with them so that they give you the clues for what is to be said in the dark. Whether regular size, or 2.0 by 2.0 slides are used, be certain to use the masks that are standard for that size.

Mark slides clearly with a sticker in the upper right hand corner of the slide as it is to be put into the projector. Number your slides so that if their order is disturbed for any reason the slides can be quickly identified. The paper you are reading should have the numbers of the slides in the proper place for ready reference if necessary.

Give your slides to the projectionist in a proper container with the slides arranged in the slots in the order they are to be projected.

Don't ask the projectionist to go back to a slide previously projected. It is much better to have a duplicate slide made.

Be certain that the slides are clean; fingerprints on the slides add nothing to your paper.

If you are using lettered diagrams, have the lettering large enough so that the person in the last row can read is easily.

If a photograph of typing is employed, see that a new ribbon is used and that the slide does not have more than 10 double-spaced lines. If the man in the last row can't read it, the slide is wasted, and it will help you to "lose your audience."

It is important that your slides be prepared long enough in advance that a "retake" is possible, if indicated.

Speak to the man in the last row. Don't "mug the microphone." Disregard it entirely. It is set up with this in mind.

Use your slides. Point out to the audience the points you are attempting to show. Remember, your audience has not seen the slide and therefore it is not familiar with it.

If, when facing the screen to demonstrate the slide, you do not have a neck microphone, speak louder so that you can be heard.

In a 15-minute paper, it is not necessary to repeat a conclusion. If your audience doesn't remember what you have said during that time, a repeated conclusion won't help.

Close your paper with a "punch line" as is done in a play so that your audience knows when you are finished. This can be a statement such as: It is hoped this presentation has brought out (whatever you have been trying to present).

Remember, you are asking your friends to listen to your paper. It is important that you do everything you can to make it a pleasure for them to listen. The individuals who make the best presentations are the ones who have prepared the paper carefully and who have worked on it long enough to be familiar with what they are to say and also know how long it will take to say it.

Frederick C. Cordes.

PROSPECTS IN THE PREVENTION OF BLINDNESS*

The comprehensive welfare services for the blind in this country have yielded much statistical information. Sorsby's reports[1-3] have been supplemented by his summaries in the annual reports of the chief medical officer of the Ministry of Health.[4] An outstanding problem revealed by these studies was the high proportion of cataract patients—about 25 percent—amongst those registered as blind. Some 80 percent of those registered as blind from cataract had not sought or obtained treatment. In 1954 the Ministry of Health made available more beds for cataract patients and sought to distribute the waiting-lists more evenly,[5] and now it has issued a

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* Reprinted from *The Lancet*, March 15, 1958.

direct appeal to general practitioners, who in the first place have charge of the potential cataract patient. A widely distributed memorandum[6] points out that the old teaching that operation could not be undertaken until the cataract was ripe is no longer valid, for by modern methods the cataract can be removed as soon as vision is seriously reduced. One difficulty is that no less than a third of new registrations of blindness relate to people over the age of 80 years; and, though cataract surgery at such an advanced age generally presents no special problems, in many patients operation might have been undertaken under better conditions at an earlier age. It is disturbing that, in spite of the general medical services available to all under the National Health Service and the recent expansion of ophthalmic departments, most of those seeking admission to the Blind Register apply through the National Assistance Board, and not through medical agencies.

Cataract is generally regarded as a "senile" affection, and so are the macular lesions misnamed senile macular degeneration. Together these two affections account for nearly 50 percent of all registrations of blindness. But out of altogether nearly five million aged over 65 years only some several thousands a year are affected by these lesions; the Ministry's memorandum rightly emphasizes that old age by itself does not cause blindness, and the nature of these two affections is unknown. Little is known, either, of glaucoma, another major cause of blindness, which tends to affect younger patients. Though much can be hoped for from administrative action on cataract, there is little immediate prospect of reducing the incidence of blindness from senile macular degeneration or from glaucoma, but early diagnosis and treatment are essential in the management of glaucoma.[7]

Over-all surveys—because of the high proportion of aged among the newly registered—necessarily emphasize cataract, senile degeneration, and glaucoma. A different picture emerges if attention is confined to the blind under the age of 50 years. Here optic atrophy stands out as the largest single group, with 22.6 percent of the total in 1955 and 23.0 percent in 1956; and among the specified causes of optic atrophy "prenatal influences," intracranial lesions, and disseminated sclerosis were outstanding with a total of 65.8 percent, while in some 18 percent no cause was established. Optic atrophy therefore calls for urgent investigation if the amount of blindness in the age-groups under 50 is to be substantially diminished. Retinitis pigmentosa and its allied conditions, myopia, congenital defects, diabetic retinopathy, and iritis and iridocyclitis, all account for a substantial quota of cases.

Any survey of the causes of blindness today shows that there are one major surgical problem—cataract—and a mass of medical problems. Traditionally ophthalmology is largely a surgical specialty; and the recognition that a substantial series of medical problems lie before it is in itself a considerable advance. Today the general practitioner is being urged to select his cases for surgical treatment but he may be called on to play a more active part in the ophthalmology of the future.

References

1. Sorsby, A.: The causes of blindness in England and Wales. M. Research Council, memorandum No. 24, London, H. M. Stationery Office, 1950.
2. ———: The causes of blindness in England, 1948-1950: A report to the Ministry of Health. London, H. M. Stationery Office, 1953.
3. ———: Blindness in England, 1951-1954: A report to the Ministry of Health. London, H. M. Stationery Office, 1956.
4. ———: Blind certification, 1955, in the Report to the Ministry of Health, 1955, pt. II, p. 218; Blind certification, 1956, ibid, 1956, pt. II, p. 215. London, H. M. Stationery Office, 1956 and 1956.
5. Ministry of Health: Treatment of cataract and glaucoma. R. H. B. (53) 115.
6. ———: Memorandum on the prevention and alleviation of blindness.
7. Davenport, R. C.: Practitioner, **178**:562, 1957; Law, F. W.: Canadian M. A. J., **78**:1, 1958.

OBITUARIES

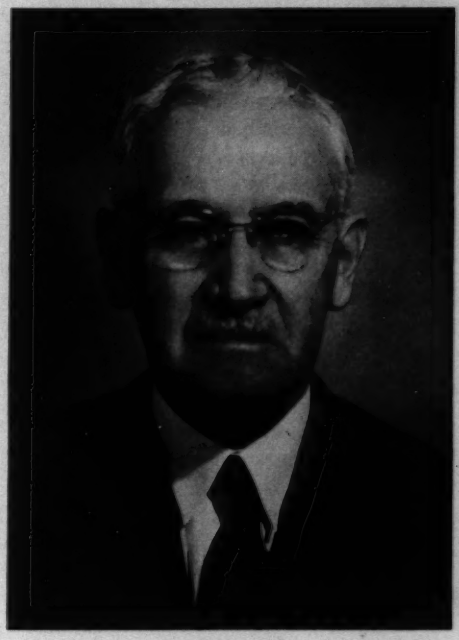
MANUEL URIBE TRONCOSO, M.D. (1867-1959)

Dr. Manuel Uribe Troncoso, international authority on ophthalmology and the beloved senior member of the editorial board of THE JOURNAL, died at his home in New York City on January 23, 1959, in the 92nd year of his life. Dr. Troncoso was the last of the chief editors of the six publications (he had founded the *Anales de Oftalmologie* in 1898) which joined forces in 1918 to form the present Series 3 of THE AMERICAN JOURNAL OF OPHTHALMOLOGY.

Manuel Uribe Troncoso was born on June 17, 1867, in the city of Toluca, capital of the state of Mexico, the son of Romualdo Uribe and Guadalupe Troncoso. He was one of 16 children. After his preparatory schooling at the Scientific and Literary Institute of the state of Mexico, where he was a distinguished pupil, he studied medicine at the University of Mexico, receiving the degree of doctor of medicine on April 15, 1890. His thesis, an original research study entitled, "A study on herpetic keratitis," foreshadowed his career as a great ophthalmologist. Two years after his graduation, Dr. Troncoso, as a member of the Section on Ophthalmology, assisted at the First Mexican Medical Congress held in Mexico City in December, 1892.

On August 17, 1893, in Mexico City, Dr. Troncoso was married to Miss Maria Alas, and to them were born seven children.

In 1898 he founded the *Anales de Oftalmologie* and, in 1899, he was named to the staff of the Ophthalmic Hospital which had been opened in Mexico City in 1898. Attending the XIII International Conference on Hygiene and Public Health in Berlin in September, 1907, he remained in Europe until 1908, studying ophthalmology and medical examinations in schools. On his return to Mexico, he organized a department of student hygiene with a staff of 21 doctors and three nurses who assisted him in the medical examination of 35,000 pupils.



MANUEL URIBE TRONCOSO, M.D.

When Dr. Troncoso moved to New York in 1916, he received from the regents of the University of the State of New York the extraordinary distinction of being granted a license to practice medicine without examination because of his "conceded eminence and authority in his profession." From 1916 on, Dr. Troncoso dedicated himself completely to ophthalmology. He became professor of ophthalmology at the Post-Graduate Medical School and Hospital of New York City. In 1932, he gave up this post to accept an appointment to the Institute of Ophthalmology of the College of Physicians and Surgeons, Columbia University, where he did research work. Until his retirement he served as assistant professor of ophthalmology at Presbyterian Hospital (Columbia University).

Dr. Troncoso's contributions to ophthalmology have been many and varied. In 1916, he founded the Spanish-American Medical Society of New York. In 1945, he invented

the gonioscope and his second book, *A Treatise on Gonioscopy*, was published in 1947 and reprinted in 1948. The first such book in the world, it marked Dr. Troncoso a pioneer in this method of eye examination. His first book, *Internal Diseases of the Eye and Atlas of Ophthalmoscopy*, was published in 1937; the second American edition appeared in 1950 and the Spanish translation was published in Mexico in 1952. Dr. Troncoso also wrote more than 150 articles on medicine, ophthalmology, and school hygiene, papers which have been published in Spanish, French, German, and English. During his long and active life, Manuel Uribe Troncoso contributed greatly to the welfare of mankind, and particularly to the progress of ophthalmology.

It will be recalled that the June, 1957 (volume 43: No. 6), issue of THE JOURNAL was dedicated to Dr. Troncoso and in it may be found a more detailed curriculum vitae.

Derrick Vail.

NORMA BERTHA ELLES, M.D.

NORMA BERTHA ELLES, M.D.
(1884-1959)

Norma Bertha Elles was born in Evansville, Indiana, June 30, 1884, daughter of Jacob and Caroline Elles, where she lived and attended public school, finishing high school in 1902. She attended the University of Michigan at Ann Arbor and received her A.B. degree and M.D. degree in 1906. Her internship was done in the old Women's Hospital of Chicago, 32nd and Rhodes Avenue.

She went to Houston, Texas, and did general practice for three years. Then, through the influence of Dr. Archer, she became interested in the study of ophthalmology. She was encouraged in her training by Dr. E. V. L. Brown in Chicago where she spent the next four years in study and a residency at the Illinois Eye and Ear Infirmary. After a year at the Sorbonne and work in Vienna she began the private practice of ophthalmology in Houston, Texas, in 1912 where she practiced continuously until her retirement in 1952. Many of her summers during those years were spent in the study of ophthalmology in Italy, Vienna, London, or Paris pursuing some special subject as refraction, slitlamp, or orthoptics.

She was a member of the A.M.A., Texas State Medical Association, Mississippi Medical Association, American Women's Medical Association, American Academy of Ophthalmology and Otolaryngology, and the American Ophthalmological Society.

At the request of Mr. and Mrs. Daniel Ripley of Houston, Texas, old friends and patients, she became interested in the organization of settlement houses and went to London to study their plans and development, and returned to form the Ripley Settlement House in Houston for the Ripley Foundation.

Dr. Elles spent the winters of 1956 and 1957 in Haiti and aided Dr. William Mellon in organizing the eye service in the Schweitzer Memorial Hospital of Haiti. An article

appeared in THE AMERICAN JOURNAL OF OPHTHALMOLOGY in June, 1958, which described her service there.

Dr. Elles retired, due to poor health, to Kalamazoo, Michigan, in 1952 to be near members of her family. She had returned to Houston for a short visit when she developed a heart attack and died on January 12th in her beloved Houston. Memorial services were held in Houston and also in Kalamazoo.

She is survived by her sister, Mrs. Hubert North, 2001 Waite Avenue, Kalamazoo, Michigan, and nieces and nephews.

Beulah Cushman.

WATSON GAILEY
(1882-1959)

I first met him as I was walking down the corridor from our suite at the Palmer House with big Jim White. He approached us from around a corner, having just come in from Bloomington, Illinois, for the academy meeting. He walked with somewhat mincing short steps, hands in pockets, whistling and wearing his homburg square upon his large head. Immediately upon recognizing Jim White his face lighted up into an impish smile and one knew immediately he had the devil just where he wanted him. Then we were introduced—so this was Dr. Watson Gailey!

We returned to our quarters and, having heard so much about his fine new clinic and excellent work, my inquiries were largely along this subject, when he said, "Come on down after the academy and see for yourself."

Well, indeed you had to see it for yourself. He had bought a city block near the business section, razed the old mansion, left the beautiful old iron fence and had built a good sized building right in the center and captioned it in modest fashion, "Gailey Eye Clinic." The outside was something to behold but the inside was even more impressive.

The interior was divided into large daylighted reception room, offices, and many examining rooms, as well as a laboratory, all completely equipped each as a unit, with push buttons, switches, and communicating telephones, designed for most efficient performance. But the finest of all his equipment was the competent staff of younger men and women he had gathered about him to carry on his work and to this end it can be said that he devoted his life to combining knowledge and skill and the means to apply it. His staff now comprises eight ophthalmologists and around 50 total personnel. Let it be told of Dr. Gailey that he shared most generously with those eager to serve.

Dr. Gailey was born September 7, 1882, at Ashland, Illinois, the son of Watson and Elizabeth Sinclair Gailey. He had a sister and three brothers, the latter having predeceased him. He married Louise Huffaker on October 29, 1908, at Jacksonville and is survived by her and a daughter, Mrs. Janet Branch, married to a Peoria surgeon, and by two grandsons.

After graduating from the College of Physicians and Surgeons of the University of Illinois he served at Cook County Hospital in 1904-1905 and at the Illinois Eye and Ear Infirmary and for the Illinois Steel Company in 1906-1907 and began practice in Bloomington in 1908.

In 1912-1913 and again in 1924 under A. Pillat he furthered his studies at the University of Vienna. Later, in 1931, he went to India in the company of the late Joseph Hompes of Lincoln, Nebraska, where in the blistering heat of Baluchistan and Afghanistan the two of them sweated it out removing myriads of cataracts. He had served as a captain in the Army Medical Corps during World War I and it was at the invitation of the British Army that he went to India.

In 1946 Dr. Gailey flew to Guatemala as consultant for an expedition of doctors and entomologists for the Pan-American Sanitary Bureau to study onchocerciasis, a common cause of blindness among the coffee workers.

Dr. Gailey lectured at many medical meet-

ings, taking part in the American Academy Instruction Course, the Mid-Winter Course in Los Angeles, and many others. He wrote some—a major contribution being the *Eye Digest* published by the Watson Gailey Eye Foundation.

Meanwhile as his volume of work and staff grew at the clinic, the Watson Gailey Eye Foundation was granted a charter of incorporation by the State of Illinois and his clinic was approved for eye residency by the American Board of Ophthalmology. In addition the Foundation pays the fees of two Illinois Normal students preparing to teach persons who are visually handicapped. It also established an eye-bank at Mennonite Hospital, working in co-operation with other eye-banks in the country.

Dr. Gailey brought great credit to himself, his relatively small community, and to ophthalmology, as a specialist and as a humanitarian. He loved and was well loved by all who knew him. In the basement of his clinic is a large assembly room with photomurals on all four walls of his pals in ophthalmology, the great, and the somewhat lesser great. He did not seek the anerobic councils of the great but that they had much in common is attested by these walls, as well as is his high regard for all his friends. Dr. Gailey was a mason, shriner, American legionnaire, and a member of A. O. A., and Phi Rho Sigma.

His death on Monday, January 19, 1959, brought to memory the beautiful home adjacent to the golf course, his wife Louise, Watson at the Hammond organ, the clinic with its eager staff, the brisk air, and the glorious coloring of fall in Central Illinois.

Harold F. Whalman.

CORRESPONDENCE

Skiascope

Editor,
American Journal of Ophthalmology:

In the August, 1958, number of The American Journal of Ophthalmology, Dr. G. Peter Halberg describes the conversion of an old DeZeng refractor into a portable device that he has found exceptionally useful.

Some years ago, at the annual meeting of the American Academy of Ophthalmology and Otolaryngology, while browsing among the commercial exhibits in ophthalmology of Mr. Henry Matalene, then of the Meyrowitz Surgical Instruments Company, I came upon a device called a skiascope. This excellent piece of equipment is made by Carl Zeiss of Jena, Germany, and consists of a double battery of lenses in a very neat paddle-shaped arrangement easily transportable in a small wooden box measuring 1.5 by 3.25 by 11 inches and weighing approximately one lb., three oz. The power of the lenses ranges from plus-20 to minus-20 diopters and the readings of the lens powers can easily be made by diverting the retinoscope beam during the actual act of retinoscopy.

The lenses are of the usual excellent quality associated with the name Carl Zeiss and proceed up and down through the dioptric limits mentioned above in half-diopter steps if necessary.

Since one of my great interests is the ophthalmic care of individuals in remote villages of Alaska and since I travel to many of these villages in my own small airplane where weight and volume are of great importance, I have found this instrument very useful. After a few weeks with one's favorite type of retinoscope and this skiascope, it is possible to prescribe spectacles on the basis of retinoscopy alone with a surprising degree of accuracy—a fact which I have ascertained by the most accurate refraction that I can do in my office here in Anchorage.

I have become so attached to this instrument and depend upon it to such a degree that at the recent meeting of the academy in Chicago, I ordered another Carl Zeiss skiascope just in case the one I have should become lost, borrowed by someone who feels he needs it more than I do, or boiled up with a pack of surgical instruments.

I offer this as a supplement to Dr. Halberg's contribution.

(Signed) Milo H. Fritz,
Anchorage, Alaska.

BOOK REVIEWS

EYE SURGERY. By H. B. Stallard. Baltimore, Maryland, Williams and Wilkins Company, 1958. Third edition, revised. 858 pages 671 illustrations, index. Price: $18.00.

H. B. Stallard, F.R.C.S., etc., surgeon to Moorfields and Barts, is well known to most of us by this time. The fact that his book on eye surgery has gone into its third revised edition in the space of a few years, attests to its popularity and value to ophthalmologists who read English. The present edition has been revised and some of it rewritten, new illustrations have been added or replace old ones, and the subject matter is brought up to date. It is natural and right that the author emphasizes the surgical procedures with which he is most familiar, or which he has himself devised with much originality. But he is very fair and, what is more important, has given us detailed descriptions and excellent illustrations in all aspects of ophthalmic surgery, so that the tyro does not have to grope or fumble. The chapter on the eyelids and reconstructive surgery is, I thought, particularly good in this respect. His chapter on cataract extraction is also worthy of note. It is interesting to see that, while he discusses the Ridley lens and its use in great detail, he remains conservative about it and carefully points out its complications, some of which are very grave indeed. However, it is unfair to pick out particular chapters for special praise, for they are all good.

Derrick Vail.

A HISTORY OF OPHTHALMOLOGY. By George E. Arrington, Jr., M.D. New York, M.D. Publications, 1959. 174 pages, bibliography, index. Price $4.00.

The series of *MD Monographs on Medical History*, edited by Martí-Ibañez, editor of the medical news magazine, *M.D.*, emphasizes the history of various specialties in relation to the history of civilization. This third volume, like the others, includes an appendix that lists the chronology of the major contributions and a directory of the current journals and societies in the special field. Consequently Arrington stresses more the relationship of ophthalmic progress to contemporary medical, biologic, and cultural concepts than factual data which are presented often but vaguely and uncritically. Thus Brisseau's name is cited twice but his precise contribution is not mentioned. Descartes is given credit for the law of refraction with no inkling of his indebtedness to Snell. Leonardo da Vinci is allotted a special chapter though his contributions were unrevealed for three centuries during which they were all in time independently rediscovered. He is on firmer ground in attributing our basic knowledge of the physiology of sight to four men—Thomas Young, Johannes Müller, Perkinje, and von Helmholtz. Purkinje actually discovered the fundus reflex (1823), but his Latin paper thereon attracted no attention and was unknown to Helmholtz. Arrington rightly contends that "The history of ophthalmology demonstrates vividly how practical application in medicine grows from knowledge of things of seemingly little value." It is to be hoped that the theme of inspirational exhortation that dominates the volume will not be lost on the younger ophthalmologists. "Man doth not live by bread only."

James E. Lebensohn.

THE EYE: A CLINICAL AND BASIC SCIENCE BOOK. By E. Howard Bedrossian, M.D. Springfield, Ill., Charles C Thomas, 1958. 352 pages, 52 illustrations, index. Price: $11.00.

This book, intended for residents and ophthalmologists preparing for the board exami-

nations, begins with a basic science section that occupies almost one-half the volume—which outlines ocular embryology, anatomy, pathology, physiology, biochemistry, and optics. Clinical refraction and surgery are not discussed. The author lapses occasionally from precise language, as when he terms the image of a plane mirror "perverted" instead of laterally inverted, and erroneously hyphenates Argyll Robertson. For no apparent reason radiational cataracts are included in the chapter on ocular emergencies. The clinical section features numerous tables of differential diagnosis including the various forms of blepharitis, keratoconjunctivitis, and optic atrophy, thyrotoxic *v.* thyrotropic exophthalmos, spasmus nutans *v.* congenital nystagmus, Adie's *v.* Argyll Robertson pupils, and the distinctions of conjunctivitis, glaucoma, and iritis. The fundus findings in normal full-term and premature infants are contrasted with the retinopathy of prematurity. Two pages in color illustrate the active and cicatricial phases of the last condition. Listed also is the Keith-Wagener interpretation of the ophthalmoscopic picture in hypertensive conditions. The therapeutic references are inadequate, and not infrequently dubious, debatable, or outdated, such as atropine for massive hyphema, miotics for central vein occlusion, and neutral ammonium tartrate for lime burns.

For passing examinations this "compend" of ophthalmology should be helpful provided its limitations are understood; for a successful career, however, unabridged texts will be of greater assistance.

James E. Lebensohn.

La Méthode Optopsychopédagogique. By G. Carlevaro and H. Ouillon. Minerva Medica. 146 pages. Price: Not listed.

This excellent book which emanates from the ophthalmologic clinic of the University of Turin is published by Minerva Medica in French. The authors provide a background that facilitates an understanding of the process of normal vision and describe their procedure of training as a means of helping a child to achieve adequate binocular vision. They also discuss the principles which underlie their therapeutic system.

The discussion of each topic is perspicuous and of welcome brevity but with an entirely adequate presentation. The text is invitingly displayed and generously illustrated.

F. H. Haessler.

Pathologie des Auges. By Prof. Magdo Radnót. Budapest, Ungarischen Akademie der Wissenschaften, 1959. Fourth edition (second in German) enlarged. 221 pages, 285 illustrations, 20 in color, references, bibliography. Price: Not listed.

American ophthalmologists are, for the most part, not familiar with the previous editions of this work published in the German first edition in 1952. The present volume is quite up to date and shows not only extensive knowledge of the author on the subject, but wide reading in the ophthalmic literature outside of the iron curtain and, what is more important, reveals excellent judgment in the selection of the world's literature of modern time. The illustrations are good, although perhaps not magnified enough to show intimate details, but the generosity of their number makes up for this in large measure. The text is adequate without being laborious. The paper and printing are of the highest quality, which I must confess comes as a surprise to me, considering the terrible times our Hungarian colleagues have endured in recent dreadful memory. Those who read German will find this book of practical value, for it emphasizes the pathologic findings with what is seen in the clinic, something that is not always done in books on eye pathology. Even if you don't read medical German, you will profit by looking at the illustrations and their captions. It is a good addition to our growing library on ocular pathology.

Derrick Vail.

ABSTRACT DEPARTMENT

EDITED BY DR. F. HERBERT HAESSLER

Abstracts are classified under the divisions listed below. It must be remembered that any given paper may belong to several divisions of ophthalmology, although here it is mentioned only in one. Not all of the headings will necessarily be found in any one issue of the Journal.

CLASSIFICATION

1. Anatomy, embryology, and comparative ophthalmology
2. General pathology, bacteriology, immunology
3. Vegetative physiology, biochemistry, pharmacology, toxicology
4. Physiologic optics, refraction, color vision
5. Diagnosis and therapy
6. Ocular motility
7. Conjunctiva, cornea, sclera
8. Uvea, sympathetic disease, aqueous
9. Glaucoma and ocular tension
10. Crystalline lens
11. Retina and vitreous
12. Optic nerve and chiasm
13. Neuro-ophthalmology
14. Eyeball, orbit, sinuses
15. Eyelids, lacrimal apparatus
16. Tumors
17. Injuries
18. Systemic disease and parasites
19. Congenital deformities, heredity
20. Hygiene, sociology, education, and history

1

ANATOMY, EMBRYOLOGY, AND COMPARATIVE OPTHALMOLOGY

Gregersen, E. **The tissue spaces in the human iris and their communication with the anterior chamber by way of the iridic crypts.** Acta ophth. **36**:819-828, 1958.

The anterior chamber of enucleated human eyes was washed with a suspension of killed cocci (diameter 0.5-1.0 μ); they penetrated deep into the vascular layer of the iris through the crypts. They could, however, not pass the much smaller openings of the iris pores. From the crypts the cocci spread far into the vascular layer, mostly through the clefts of Fuchs. Thus, the crypts and the spaces in the vascular layer make up a large-dimensioned irregular canal system. In the chamber angle the cocci were localized mostly posterior of the iris root and the ciliary body. In rabbit eyes, which have no iris crypts, the cocci did not penetrate into the iris except at the root. Human red blood cells passed into the human iris stroma through some crypts, but to a slighter degree than the cocci. (3 figures, 12 references)

John J. Stern.

Ishida, T. **A study of corneal tissue by electron microscopy.** Acta Soc. Ophth. Japan **62**:1324-1331, 2220-2227, Sept., Nov., 1958.

Sections of the corneal epithelium, basement membrane and Bowman's membrane of man and rabbit were studied by electron microscopy. In rabbits the intercellular space of the corneal epithelium is wider at the periphery of the cornea than at the center. In man such a difference is not observed. In the leucomatous cornea of man, however, the space is apt to be wider. The basement membrane is a double membrane. Bowman's membrane consists of small irregular fibrils of about 200 Å in width. (20 figures, 31 references)

Yukihiko Mitsui.

Oksala, A. and Lehtinen, A. **Studies on the structure of the vitreous body after refrigeration with carbon dioxide ice.** Acta ophth. **36**:929-939, 1958.

Bovine eyes were refrigerated with carbon dioxide ice and cut into two parts. The cut surfaces were polished to a mirror-like smoothness with very fine grinding papers. The frozen vitreous was more closely attached to the bulbar wall at the sites of the ciliary body, equator and optic disc, and more loosely near the macula. Fibers ran from the points of adhesion in two directions—to the center of the

vitreous towards Cloquet's canal and towards the next point of adhesion. The zonula formed a group of fibers which was distinct from the rest of the vitreous. Microscopic examination showed a netlike framework composed of fibers which were finer in the center but formed larger meshes. (4 figures, 41 references)

John J. Stern.

2

GENERAL PATHOLOGY, BACTERIOLOGY, IMMUNOLOGY

Fazakas, S. **Review of secondary mycoses associated with diseases of lid margins, conjunctiva and cornea.** Szemeszet 95:128-135, Sept., 1958.

The author discusses the results of his mycologic investigations initiated several decades ago, including the study of the fungus flora in healthy eyes and those with nonmycotic diseases. The parasitic nature of fungi believed earlier to be only saprophytes has been demonstrated. The parasitic varieties of these fungi display, in vivo and in vitro, the same behavior as the fungi considered parasitic previously. In cases in which the fungus played the role of a direct pathogenic agent, the view of the author has been confirmed by other published data. The question whether there are secondary mycoses in the eye as in the skin he answers in the affirmative on the base of simultaneous bacterium or virus-induced diseases and the oculomycoses associated with the former. Gyula Lugossy.

Kurisaki, M. and Kimura, R. **Experimental studies on ocular toxoplasmosis.** Acta Soc. Ophth. Japan 62:2394-2398, Dec. 1958.

From the second to the eighth day after inoculation of the vitreous of rabbits with toxoplasma, a severe iridocyclitis accompanied by corneal opacity, hyphema and subconjunctival infiltration occurred. A few days thereafter the animals died of systemic infection. In the iris and ciliary body a definite infiltration with lymphocytes and histiocytes was demonstrated histologically. Hemorrhage was also obvious. The tissue was apt to become necrotic. The inflammation extended to other parts of the eyeball and the organism was demonstrated from every part of the eye. One exceptional rabbit survived for 23 days. In this animal a granulomatous inflammation was histologically demonstrable in the retina. The methylene blue dye test of the aqueous was negative in all of the animals. (6 figures, 4 tables, 8 references) Yukihiko Mitsui.

Okámoto, I., Masuda, K. and Kobayashi, S. **Corneal immunity in vaccinia keratitis.** Acta Soc. Ophth. Japan 62:1353-1358, Sept., 1958.

Vaccinia keratitis is brought about in rabbits by the technique described elsewhere. The corneal extract is prepared after various intervals and examined for neutralizing antibody to vaccinia virus by a tissue culture procedure. A definite rise in the specific neutralizing antibody is demonstrated in the corneal extract. By intradermal vaccination a corneal immunity also results, but of slight degree. The ratio of corneal antibody titer to serum titer is 1.5:1 by corneal vaccination, and 1:28 by dermal vaccination. However, when nonspecific keratitis is brought about by an intracorneal injection of turpentine, a greater corneal immunity is obtained by intradermal vaccination; the ratio of the corneal to serum titer reaches 1:6. An administration of hydrocortisone has no effect on the antibody formation. (3 figures, 2 tables, 16 references)

Yukihiko Mitsui.

Syverton, J. T., Lennette, E. H. and Thygeson, P. **Symposium: Viruses and viral diseases.** Tr. Am. Acad. Ophth. 62: 349-431, May-June, 1958.

Syverton, Jerome T. **1957 status and prospects.** pp. 394-398.

A virus is defined as a nucleoprotein particle that is able to induce its own replication by a susceptible host cell on which it is completely dependent and to which it is essentially foreign. This process is detrimental to the host cell, either through a toxic effect of the replicated virus or to inability of the host to sustain both lives. The true virus is a microorganism so degraded by stripping that nothing remains but a genetic mechanism to ensure replication and a protein package for its protection and deliverance. It consists of a folded nucleoprotein chain enclosed in specific protein.

Recent advances and study have occurred through the derivation of strains of free living animal cells that can be kept in stable continuous culture, and means for dispersing them into suspension, and adaptation into a monolayer form for continuous growth on glass. Cell culture techniques allow for rapid and precise isolation and identification of viruses. Through this, more human disease entities can be recognized and studied. Through continued studies, possibly synthetic viruses may be developed, which can be introduced into the host to block the virulent viruses. (18 references)

Lennette, Edwin H. **Viral infections of the upper respiratory tract.** pp. 399-410.

Not abstracted.

Thygeson, Phillips. **Viral infections of the eye and adnexa.** pp. 411-431.

The specific viruses of significance in the eye are discussed individually; 29 entities are listed in this comprehensive paper.

Trachoma should be recognized by its conjunctival follicles over the upper tarsal plate, upper limbal vascularization, epithelial keratitis, subepithelial infiltrates, limbal follicles and Herbert's pits (the cicatricial remains of limbal follicles) Treatment should be continued for at least three weeks. In the undeveloped endemic areas, long-acting Kynex appears to hold promise. Laboratory aids are the presence of inclusion bodies in the early stages, and Leber cells and plasma cells in later stages.

Inclusion body keratitis is becoming less common as its venereal source is being suppressed by antibiotics. Lymphogranuloma venereum also is becoming less common for the same reason, but it should be considered, and the Frei test used when in doubt.

Conjunctivitis is often the presenting symptom of the increasingly important pharyngoconjunctival fever. Keratitis may occur in this condition, but is much shorter in duration than that of epidemic keratoconjunctivitis. In epidemics the disease usually affects children, but sporadic cases are seen in young adults.

The distinctive keratitis of epidemic keratoconjunctivitis is described. About a third of patients have pseudomembranes. The infiltrates may last for months. Adults are affected predominantly; and there is evidence that adenovirus type 8 is the cause.

Newcastle disease causes conjunctivitis without keratitis among animal handlers in this country; Orf causes lid lesions among sheep handlers in Australia; and Rift Valley fever causes chorioretinal lesions among farmers in Africa. Foot and mouth disease also may cause keratoconjunctivitis.

Molluscum contagiosum and verruca vulgaris occur on the lid margins. The former usually causes a follicular conjunctivitis and if undiagnosed this may go on to pannus. Verruca may cause a nonfollicular conjunctivitis, but usually causes keratitis. Removal of the lip lesions cures the membranal lesions.

The commonest cause of keratoconjunctivitis in man is measles. This rarely leaves serious sequelae and may be found early in practically every case. Mumps

may cause an interstitial keratitis. Varicella rarely causes vesicles on the eyelids, but may occur also on the conjunctiva and cornea. Variola still occurs, and vaccinia is often transferred to the eyelids. Hyperimmune vaccinia gamma globulin may be of dramatic value. Herpes Zoster ophthalmicus is in this group and is becoming more important as our life span increases. It may cause exudative anterior choroiditis resulting in retinal detachment. Glaucoma is an all too frequent development.

Herpes simplex is still the most important type of keratitis in the United States. The dendrites are difficult to detect in infants and young children. Herpes is divided into superficial and deep lesions. The former includes dendritic, punctate, filamentary, striate, vesicular, geographic, neuroparalytic and chronic epithelial subtypes. The latter includes disciform, diffuse interstitial ulcerative with hypopyon, and keratouveitis. The virus now is much easier to identify in chorioallantoic membrane than on rabbit's cornea. In conjunctival scrapings the cellular reaction is mononuclear. In corneal scrapings characteristic giant multinuclear epithelial cells are seen and are useful in diagnosis because the only other viruses which produce this type of epithelial change give rise to clinically recognizable diagnostic lesions which exclude them from consideration. The many trigger mechanisms such as fever, sunlight, and emotional strain, are enumerated and must be avoided. The use of cortisone, of course, is condemned.

Cytomegalic inclusion disease is a congenital and intrauterine infection which may cause chorioretinitis. Maternal rubella in the first trimester may cause cataract and other deformities.

Poliomyelitis may cause optic neuritis or motor nerve paralysis, as may other viruses which cause encephalitis.

Beal's conjunctivitis is probably pharyngoconjunctival fever. In superficial punctate keratitis the presence of a causative virus has not yet been confirmed, nor has it been in sympathetic ophthalmia. Behçet's disease, although rare here, is important because it invariably causes blindness. Cat scratch granuloma, the conjunctivitis of epidemic hemorrhagic fever, and chronic follicular conjunctivitis still must await confirmation of a viral etiology. Several unidentified cases of keratitis and keratoconjunctivitis have been studied and work may yet reveal further viral entities in such cases.

A clear plea is made for adequate modern sterilization techniques to avoid passing on these viruses in the doctor's office and the hospital and also to avoid viral hepatitis. (24 figures, 44 references)

Harry Horwich.

3

VEGETATIVE PHYSIOLOGY, BIOCHEMISTRY, PHARMACOLOGY, TOXICOLOGY

Bettman, J. W., Fellows, V., Chao, P. and Johnson, J. P. **The effect of tonography and other pressures on the intraocular blood volume.** A.M.A. Arch. Ophth. **60**:230-236, Aug., 1958.

These studies indicate that the volume of blood in an eye is very much less than the volume of aqueous. Tonometry changes the blood volume but little, but with an orbitonometer the intraocular blood volume is markedly reduced. (3 figures, 2 tables, 24 references)

G. S. Tyner.

De Berardinis, E. **Chemical chromatographic separation of the phosphorated components present in the acid-soluble fraction of bovine retina.** Acta ophth. **36**: 807-814, 1958.

A new chemical and chromatographical technique for demonstrating phosphorated compounds confirmed the existence in the retina of an active glucose metabolism which follows normal paths but could

not exclude a possible collateral route. (3 figures, 2 tables, 17 references)

John J. Stern.

De Conciliis, U. and De Simone, S. **The influence of butazolidine on lenticular ATP.** Arch. di ottal. 62:317-320, July-Aug., 1958.

Butazolidine disturbs the phosphorylation of corneal tissue in vitro, particularly to change the concentration of ADP and ATP. Rabbits treated for 30 days with 150 mg./kg intramuscularly showed no effect on lens ATP. (1 table, 4 references)

Paul W. Miles.

De Rosa, L. and Testa, M. **Activity of transaminase in the retina during experimental iodate pigmentary degeneration.** Arch. di ottal. 62:261-267, July-Aug., 1958.

Intravenous sodium iodate in rabbits caused congestion of the choroid, posterior edema of the retina, disturbance of pigment epithelium, migration of pigment, and degeneration of rods and cones. Transaminase content of the retinas, determined by the method of Tonhazy, appeared unrelated to the observed retinal damage. (2 tables, 13 references)

Paul W. Miles.

De Rosa, L. and Testa, M. **Electrophoretic studies of water soluble proteins in vitreous during experimental pigmentary degeneration of the retina.** Arch. di ottal. 62:339-341, July-Aug., 1958.

Electrophoresis proteinograms indicated that the vitreous of rabbits treated with intravenous sodium iodate contained three soluble protein fractions instead of two. (2 figures, 3 references)

Paul W. Miles.

Ericson, L. A. **Twenty-four hourly variations of the aqueous flow. Examination with perilimbal suction cup.** Acta ophth. Suppl. 50, 1958.

The intraocular pressure is complex, depending on inflow, resistance to outflow, and episcleral venous pressure. The regulation of the pressure in the eye can be understood better if these factors are examined separately. In this investigation an attempt is made to answer the question whether aqueous inflow in normal eyes shows 24-hourly variation paralleling the 24-hour variation of intraocular pressure. Furthermore, an attempt is made to demonstrate the influence of sleep in the aqueous inflow of persons working during the night.

After an extensive survey of previously published investigations the method used in the present study is described. A suction cup is applied at the limbus which compresses the outflow channels. The increase in pressure thus obtained allows estimation of the inflow. Two chapters are devoted to the sources of error and their avoidance.

The aqueous inflow was studied by examining 50 normal subjects every four hours for three days and nights. During intraocular pressure and the inflow are found. Between 8 p.m. and midnight a considerable reduction takes place, between midnight and 4 a.m. the level is constant, and between 4 a.m. and 8 the level of the day value is regained. In 10 subjects working during the night the difference between day and night was less pronounced.

In persons working in daytime, both the intraocular pressure and the inflow are on a constant level between 8 a.m. and noon. The intraocular pressure then decreases continuously until midnight by about 3 mm. Hg. No corresponding reduction of the inflow was found during the day. Between 8 p.m. and midnight there is a considerable reduction in the inflow. Between midnight and 4 a.m. there is an increase in pressure but no changing of the inflow. Between 4 and 8 both factors increase, the inflow increasing much more than pressure. The reduction of the inflow during the night is greater

than the reduction of the intraocular pressure. This discrepancy could be explained by an increase of outflow resistance.

In subjects working in daytime there was no difference in the inflow between 8 and midnight regardless whether the subject was asleep or awake. In subjects working at night there was a reduction of inflow during the night but considerably less than that of day personnel. This reduced inflow was present until 8 when the night personnel went to bed. At noon, after four hours of sleep, the value had regained its day level. This shows that in night workers there is still a certain 24-hour rhythm but that it is reduced as compared with subjects working during the day. (8 figures, 23 tables, 199 references) John J. Stern.

François, J. and Rabaey, M. **Permeability of the capsule for the lens proteins.** Acta ophth. **36**:837-844, 1958.

The micro-electrophoresis on agar of the lens proteins shows, from the anode to the cathode, two or more rapid fractions, the large fraction of α-crystalline, various medium fractions, and three slow fractions (T_1, T_2, and T_3). The study of the permeability of the capsule for the lens proteins shows that it is not permeable for α-crystalline (high molecular weight) nor for the rapid fractions, but highly permeable for the slow fraction T_1 and T_2. These fractions are embryonal and have a very low molecular weight. The capsule is also highly permeable for one medium fraction, the most basic M_1. (Author's summary) (4 figures, 11 references) John J. Stern.

Funatsu, H. **Influence of vitamin B_2 on the respiration of rabbit cornea.** Acta Soc. Ophth. Japan **62**:1668-1682, Sept., 1958.

When rabbit cornea is dipped into a solution of flavine-adenine-dinucleotide a considerable rise in the oxygen consumption takes place when measured by Warburg's procedure. When the agent is injected subconjunctivally in as great a dose as 300γ the same effect is observed in vivo also. A small dose of 30γ has no effect. Funatsu considers that a subconjunctival injection of the agent in large doses may be clinically useful in some conditions. (7 figures, 9 tables, 53 references) Yukihiko Mitsui.

Goto, Y. **Removal of the cervical ganglion and the vitamin B_1 content of the eye.** Acta Soc. Ophth. Japan **62**:1632-1644, Sept., 1958.

After removal of the cervical ganglion the vitamin B_1 in ocular tissues usually increases. It is due to an enormous increase in co-carboxylase. The free aneurin rather reduces it. The total vitamin content in blood does not show a definite change but the co-carboxylase is apt to increase while free aneurin is reduced. (6 tables, 50 references) Yukihiko Mitsui.

Grant, W. Morton. **Ophthalmic pharmacology and toxicology.** A.M.A. Arch. Ophth. **60**:324-349, Aug., 1958.

Grant provides a most helpful review of the literature for the period April, 1957, to March, 1958. (161 references) G. S. Tyner.

Gräsbeck, R. and Takki-Luukkainen, I.-T. **Vitamin B_{12}-binding substance in human tear fluid.** Acta ophth. **36**:860-864, 1958.

Gastric juice, saliva, serum, and other biological fluids are able to form large molecular complexes with vitamin B_{12}. In electrophoretic experiments the authors showed that the tears have a similar capacity to combine with vitamin B_{12}, which was localized in one single electrophoretic fraction, probably a protein but not lysozyme. (2 figures, 14 references) John J. Stern.

Harris, J. E., Rowell, P. P. and Beaudreau, O. **The adaptation of Virac, a new iodophore, to clinical use.** A.M.A. Arch. Ophth. **60**:206-214, Aug., 1958.

Virac is an iodophore, a mixture of elemental iodine and a cationic detergent. It is less toxic than benzalkonium chloride. In germicidal strength it was not toxic to the human eye. It appears to be a superior solution for irrigation of the conjunctival tac and lacrimal passages. Virac does not cure herpes simplex. (4 figures, 5 tables, 10 references) G. S. Tyner.

Heck, J. and Zetterström, B. **Analysis of the flicker electroretinogram in the newborn.** Ophthalmologica **135**:205-210, March, 1958.

The flicker ERG was recorded in 45 normal infants 14 hours to three and one-half months of age. During the first two months a process of development was demonstrable in the ERG although all of the retina except the macula is histolgically mature at birth. Soon after birth a maximal flicker fusion frequency (FFF) of 20/sec. can be recorded and the ERG shows slower monophasic flicker waves like those of the scotopic flicker ERG of the adult. The positive off-effect suggests that photopic elements are active at 14 hours of age. The photopic potentials and the FFF have adult value at two months of age whereas the scotopic b-wave approximates adult form and magnitude at the end of the first year of life. (9 references) F. H. Haessler.

Hirose, K. and Kiya, K. **Experimental studies on the fundus changes caused by stenosis of the ascending aorta.** Acta Soc. Ophth. Japan **62**:1739-1746, Oct., 1958.

This is an experimental reproduction of "pulseless disease of Takayasu-Onishi" in rabbits. By a mechanical stenosis of the ascending aorta in infant rabbits, an arteriovenous anastomosis like that seen in the "pulseless disease" in man gradually develops in the fundus. (12 figures) Yukihiko Mitsui.

Holwich, F. **The influence of light on the change of color in the frog.** Klin. Monatsbl. f. Augenh. **133**:784-787, 1958.

The results of the experiments performed by Giersberg were duplicated. If the visual centers in the optic lobes are destroyed the frogs will be blind (no nystagmus on the revolving drum) and yet they will respond as usual by change of color when exposed to light. (2 figures, 7 references) Frederick C. Blodi.

Hoshina, M. and Konishi, K. **The influence of anoxia on the eye: development of cataract.** Acta Soc. Ophth. Japan **62**: 1413-1422, Sept., 1958.

In rats which were exposed to a low atmospheric pressure for two hours the mitosis of the lens epithelium was reduced by 56 percent in one hour and it took nine hours to regain the normal level. The oxygen consumption was reduced by 43 percent during the exposure and the glycolysis by 23 percent. By an administration of KCN, the mitosis was increased by 18 percent, the oxygen consumption decreased by 19 percent, and the glycolysis by 53 percent. After an administration of NaF, the mitosis remained unchanged, the oxygen consumption became unmeasurably low, and the glycolysis reduced by 54 percent. Among these factors it is only the low pressure that causes cataract. The authors think that the development of cataract is more closely related to the reduction in mitosis than to a change in the metabolism of the lens epithelium. (8 figures, 8 tables, 19 references) Yukihiko Mitsui.

Huggert, A. and Odeblad, E. **Studies on the water of the crystalline lens.** Acta ophth. **36**:885-890, 1958.

Exchange experiments with heavy water in vitro on cattle lenses between the

sixth fetal month and the 14th year of age showed a rapid exchange (1 to 2 hours) of 80 to 90 percent of the water content of the cortex, and almost complete exchange in 5 to 20 hours of the water content of the nucleus, depending on the age. (4 figures, 14 references)

John J. Stern.

Jacobson, J. H. and Gestring, G. F. **Centrifugal influence upon the electroretinogram.** A.M.A. Arch. Ophth. **60**:295-302, Aug., 1958.

By means of the ERG evidence is presented for the existence of centrifugal fibers in the optic nerve, a subject of interest and discussion for many years. (11 figures, 21 references) G. S. Tyner.

Kobayashi, A. **Phosphorus metabolism in the optic nerve of the dog.** Acta Soc. Ophth. Japan **62**:1582-1600, Sept., 1958.

This is a follow-up study of spinal fluid circulation by the use of radioactive phosphorus. The P^{32} is introduced into the cisterna magna of dogs. The phosphorus in the optic nerve is first determined in normal dogs. Then that in dogs with "talcum arachnoiditis" is studied. In the latter the phosphorus content is considerably lower than in the former showing a decreased circulation of spinal fluid. Then the optic nerve is removed from normal dogs and from dogs with "talcum arachnoiditis." The nerve is dipped for one hour into Krebs-Ringer phosphate buffer solution containing P^{32}. Then the nerve is measured for the content of lipoid-P and residual-P. Their concentration is low in arachnoiditis dogs. Kobayashi concludes that the low content of phosphorus in the arachnoiditis optic nerve is not only due to a disturbed circulation of the spinal fluid but also to a disturbance in the tissue metabolism. (1 figure, 8 tables, 71 references) Yukihiko Mitsui.

Marretta, P. V. and Garzino, A. **Ophthalmologic observations of a muscle-relaxing drug with central action.** Rassegna ital. d'ottal. **27**:311-319, July-Aug., 1958.

The earliest experiments with muscle-relaxing drugs is discussed. These preceded the use of curare in ophthalmic surgery. A study was recently made on 45 individuals between the ages of 21 and 34 years who had various pathologic changes in the eye. A preparation called Miorelax (an alpha-glycerol-guaiacol ether) was injected intravenously. Observations were made repeatedly for an hour. The following results were recorded: the motility of the extrinsic ocular muscle was reduced, ptosis of the upper eyelids followed, ocular tension was barely affected, modifications of the retinal blood vessels of the musculature, and slight exophthalmos. These changes indicate that the drug brings about a paresis of the striated muscle of the visual apparatus. (1 figure, 11 references) E. M. Blake.

McEwen, W. K. **Application of Poiseuille's law to aqueous outflow.** A.M.A. Arch. Ophth. **60**:290-294, Aug., 1958.

The application of Poiseuille's law to the outflow of aqueous humor leads to several interesting conclusions. The major resistance to outflow of aqueous is a tissue barrier of low porosity, averaging about one hole (2μ in diameter) every 0.01 mm^2. A decrease in the size of the holes can explain "open angle" glaucoma. A single patent hole 12μ in diameter will permit a normal facility of outflow. (1 figure, 18 references) G. S. Tyner.

Mizukawa, T. and Uyama, S. **Zone electrophoretic studies on the soluble proteins of the crystalline lens.** Jap. J. Ophth. **2**: 307-313, Oct.-Dec., 1958.

The separation of soluble lens proteins was studied throughout each stage of cataract in bovine and rabbit eyes. The α-crystalline of the cortex and nucleus have the same isoelectric point. The pro-

teins separated from the nucleus and cortex of the rabbit and bovine cataracts show the same electrical behavior. In naphthalene cataract beta crystalline decreases as the cataract develops and it disappears when the cataract is mature. (4 figures, 5 references)

Irwin E. Gaynon.

Mizukawa, T., Takagi, Y., Kiuchi, K. and Okamura, H. **Developmental mechanism of napthalene cataract in rabbits.** Acta Soc. Ophth. Japan **62**:1401-1409, Sept., 1958.

Napthalene is given to rabbits. For control, 2-naphthol and bromobenzene, both of which follow a metabolic course similar to that of naphthalene but have no cataractogenic effect, are used. The conjugated glucuronic acid in the blood is increased by any of the three agents; however, in the aqueous it is increased only by naphthalene. The free glucuronic acid, on the other hand, is increased in the aqueous only by the two control agents, and not by naphthalene. The authors suppose that the appearance of conjugated glucuronic acid in the aqueous may have a role in the development of naphthalene cataract. (16 figures, 17 references)

Yukihiko Mitsui.

Nonaka, K. **Corneal metabolism as an index for heterokeratoplasty.** Acta Soc. Ophth. Japan **62**:1348-1352, Sept., 1958.

Nonaka reports an examination of corneal metabolism to find suitable corneas for heterokeratoplasty. The metabolism of corneas from various animals is measured by Warburg's method. Chicken cornea showed the greatest oxygen consumption and glycolysis. For this reason Nonaka considers the chicken cornea to be the best for hetero-keratoplasty. (1 table, 18 references) Yukihiko Mitsui.

Prijot, E. **The rigidity of the human eye.** Acta ophth. **36**:865-873, 1958.

The ocular rigidity coefficient of Friedenwald, measured in the enucleated human eye two to six hours afer death, is not a constant. It diminishes with increasing intraocular pressure. The relationship between the rigidity coefficient and intraocular pressure varies in different species. The distension of the enucleated human eyeball does not follow Hooke's law. The rigidity of the enucleated human eye, two to six hours after death, is of the same order as that obtained in vivo with the Schiøtz tonometer. This agreement of in vivo and post mortem-values is better when McBain's tables, rather than those of Friedenwald, are used. (4 figures, 31 references) John J. Stern.

Shimooku, M. **Connections of the trigeminal and the facial nerve to the oculomotor nucleus in cats.** Acta Soc. Ophth. Japan **62**:1719-1728, Oct., 1958.

A concentric needle electrode is inserted into the nucleus of the oculomotor nerve in cats. The action potential is recorded giving an antidromic stimulation. Then the possible connection to the trigeminal and facial nerve is studied as follows. An electric stimulation of these two nerves does not cause the appearance of an action current from the oculomotor nucleus. However, when an antidromic stimulation is given to the oculomotor nerve immediately after the stimulation of one of the two nerves, the action current from the oculomotor nucleus becomes lower than that obtained without the prestimulation. When the labyrinth is destroyed, such an effect of "prestimulation" disappears from the first and third branch of trigeminal nerve, but not from the second branch of the trigeminus and the facial nerve. (3 figures, 13 references)

Yukihiko Mitsui.

Tanaka, K. **Influence of stress on the mitosis of the corneal epithelium in rabbits.** Acta Soc. Ophth. Japan **62**:1332-1347, Sept., 1958.

Electric shock produces a transient opacity in the cornea of rabbits. The mitosis of the corneal epithelium is reduced immediately after the shock. It increases four hours later and eight hours later there is again a reduction. It takes 24 hours to regain the normal state. A shock by an intravenous injection of epinephrine has a similar effect. (11 figures, 22 tables, 31 references) Yukihiko Mitsui.

Testa, M. and De Conciliis, U. **Lenticular transaminase in rabbits with experimental anaphylactic uveitis.** Arch. di ottal. 62:301-307, July-Aug., 1958.

Small amounts of the enzyme transaminase were found in lenses with early cataract, which gradually disappeared when opacity became complete.

Paul W. Miles.

Urata, Bobuyasu. **Experimental studies on the oculomotor centers.** Jap. J. Ophth. 2:284-288, Oct.-Dec., 1958.

The lateral hypothalamus and nucleus proprius of the cerebral peduncle play a part in the central mechanism of ocular movements. On increasing the intensity of the stimulus, monocular movements became binocular. Horizontal movements were all directed to the contralateral side. Vertical movements were all directed upward. No downward movement was observed. Where a difference in movement between the two eyes was observed the homolateral movement was always stronger. (1 figure, 1 table, 7 references)

Irwin E. Gaynon.

Vilmar, K.-F. and Buchmann, H. H. **A new mydriatic and its action on the pupillary musculature.** Ophthalmologica 135:114-122, Feb., 1958.

Two new mydriatic drugs (Ro 1-6272 and Ro 1-7683) are described which have the advantage that they produce adequate mydriasis without great danger of giving rise to an increase of ocular tension. The mydriasis begins to subside in an hour and this can be hastened by miotics. (3 figures, 3 tables, 5 references)

F. H. Haessler.

Yamaji, R., Muraji, Y., Matsumoto, T. and Muraji, K. **A study of the effect of amino acids on the regeneration of the visual purple.** Jap. J. Ophth. 2:273-277, Oct.-Dec., 1958.

In testing the effects of amino acids on the regeneration of visual purple in the bullfrog, it was found that phenylanine, tyrosine, alanine, valine, and isoleucine augmented remarkably the regeneration of visual purple; leucine and ornithine did so to a lesser degree; and histidine, arginine, serine, glycine and glutamic acid had a slight effect. (1 table, 12 references)

Irwin E. Gaynon.

Yoshizawa, Teruko. **Biochemical studies on the plasmoid aqueous. Report III. Nonprotein nitrogen and free amino acids.** Jap. J. Ophth. 2:300-307, Oct.-Dec., 1958.

There is a decrease in the nonprotein nitrogen in the plasmoid aqueous after the withdrawal of aqueous fluid. Immediately after withdrawal the amino acid content approaches that of the serum, and this is followed by a selective permeability for the amino acids. (4 figures, 1 table, 10 references) Irwin E. Gaynon.

4

PHYSIOLOGIC OPTICS, REFRACTION, COLOR VISION

Amano, K. **Measurement of the axial length in the living eye by X-ray photography.** Acta Soc. Ophth. Japan 62:1828-1840, Oct., 1958.

There is a definite elongation of the axial length of the eye in high myopia. However, in cases of slight myopia the relation between the axial length and the refractive power is not always parallel, indicating that slight myopia is of refractive origin and not of axial. The myopic

fundus changes can be seen when the axial length exceeds 27 mm. (5 figures, 1 table, 25 references)

Yukihiko Mitsui.

Colenbrander, M. C. **Localization.** Ophthalmologica **135**:246-250, April, 1958.

Colenbrander invents a conversation between himself and his image in a reflecting sphere in his garden which illuminates the psychologic process of the concept of space. (4 references)

F. H. Haessler.

François, J. and Verriest, G. **The relation between visual acuity and illumination of the background in subjects with normal and congenitally anomalous vision.** Ophthalmologica **135**:193-204, March, 1958.

The authors investigated the relation between the visual acuity for black optotypes and the illumination of the background in subjects with normal trichromatic vision, protanomaly, protanopia, deuteranopia, rodmonochromatism, and essential hemeralopia. In white light the mean threshold of the color-deficient subject was higher than normal.

In red light the deuteranomolous subjects have lower mean thresholds than the normal, the deuteranopes have normal thresholds and monochromatic subjects have highly pathologic thresholds.

In green and blue light the thresholds of the monochromats are lower than the normal and the mean thresholds of all groups of partial color deficiency are higher. The rise is maximal in deuteranopia. (2 figures, 3 tables, 8 references)

F. H. Haessler.

Grzibek, P. M. and Sachsenweger, R. **Experimental investigations on the objective determination of the visual acuity at the Romberg apparatus.** Klin. Monatsbl. f. Augenh. **133**:835-846, 1958.

The apparatus uses opticokinetic nystagmus for the determination of the visual acuity. There is frequently a great discrepancy between the subjective and objective visual acuity. The brightness of the background, the rotatory speed, and adaptation do not influence the results if extreme values are avoided. Fatigue, lack of attention, and other factors account for these discrepancies. (8 figures, 11 references)

Frederick C. Blodi.

Harada, M. and Hayashi, S. **Differential diagnosis of amblyopia.** Jap. J. Ophth. **2**: 268-273, Oct.-Dec., 1958.

Patients with pathologic amblyopia have absolute scotoma and decreased vision due to organic disease or high refractive error of the affected eye. In amblyopia ex anopsia there is an inability to fix in the central portion of the eye, scotoma is not present, and there is rarely organic disease or a refractive error of the eye. Strabismus may result, and the vision is less than one sixth. (2 figures, 3 tables, 5 references)

Irwin E. Gaynon.

Kato, K. and Tabata, S. **Comparison of the hue discrimination between both eyes.** Jap. J. Ophth. **2**:254-263, Oct.-Dec., 1958.

A modified Nagel's anomaloscope is used to measure the discrimination threshold for each wave length. In normal persons, and those with deuteranopia, protanopia, and protanomaly no definite difference in the discrimination limen is found between the eyes throughout the spectrum. In those with deuteranomaly a definite difference in the discrimination limen is demonstrated between the eyes in all cases. (6 figures, 5 tables, 2 references)

Irwin E. Gaynon.

Sbordone, G. **Ocular refraction with diamox.** Arch. di ottal. **62**:257-260, July-Aug., 1958.

Diamox produces no appreciable change in ocular refraction. (7 references)

Paul W. Miles.

Smejkal, V. **The possibility of surgical treatment of astigmatism.** Ophthalmologica **135**:211-222, March, 1958.

By means of experiments with rabbits the author shows that corneal scars produced by galvanocautery bring about aplanation of contiguous areas and that it is possible to adjust the corneal curvature by the application of radial linear scars. (2 figures, 2 tables, 7 references)

F. H. Haessler.

Walter, Rudolf. **Supplementary trial cases.** Klin. Monatsbl. f. Augenh. **133**:899-901, 1958.

The following are recommended: a set of prisms, a case of cataract lenses and a choice of bifocal lenses.

Frederick C. Blodi.

Yamamori, Akira. **Optical investigation of limit of visual acuity. (Report I)** Jap. J. Ophth. **2**:241-254, Oct.-Dec., 1958.

The author presents an extensive completely mathematical analysis. (13 figures, 5 tables, 2 references)

Irwin E. Gaynon.

5

DIAGNOSIS AND THERAPY

Baum, G. and Greenwood, I. **The application of ultrasonic locating techniques in ophthalmology.** A.M.A. Arch. Ophth. **60**: 263-279, Aug., 1958.

This is a preliminary report of a new method for the visualization of the interior of the opaque eye and of the retroocular areas by pulsed ultrasonic echographic methods. Foreign bodies may be localized in the presence of opaque media. Tumors in any position may be visualized. The differential diagnosis of intraocular tumors, retinal detachment, and vitreous hemorrhage is discussed. (36 figures, 3 references) G. S. Tyner.

Boberg-Ans, Jørn. **Treatment of iridocyclitis with Finsen carbon arc light.** Acta ophth. **36**:891-899, 1958.

Finsen light treatment in the form of daily light bathings for 10 to 60 minutes, for up to 86 treatments, proved valuable as in adjunct in 126 patients with chronic uveitis and iridocyclitis who failed to respond satisfactorily to cortisone, ACTH, and tuberculin. (6 figures, 9 references)

John J. Stern.

Brecher. I. **Rapid and effective disinfection of the conjunctiva for emergency operations.** Klin. Monatsbl. f. Augenh. **133**:902-904, 1958.

The author recommends a mixture of sulfathiazole and oxycyanate of mercury administered by iontophoresis. (6 references) Frederick C. Blodi.

Buxedo, Robert. **Use of piromen in ophthalmology.** A.M.A. Arch. Ophth. **60**:319-321, Aug., 1958.

Piromen is a pyrogenic substance obtained from Pseudomonas aeruginosa. The reactions are not as marked as to intravenous typhoid vaccine, nevertheless it is suspected that it stimulates antibody formation in much the same way as the vaccine. The initial dose is 5 μg. Two days later the dose is increased to 7.5 μg. and subsequent ones consist of 10 μg. (2 tables, 5 references) G. S. Tyner.

Csüllög, F. **Potentiated anesthesia in ophthalmology.** Szemeszet **95**:144-151, Dec., 1958.

The author used chlorpromazine compounds (largactil, propaphenine, prothezine, hibernal) in the preoperative management of 385 patients and describes the schedule which he uses. This pretreatment exerted a more favorable effect than the earlier phenobarbital pretreatment, as seen from the fact that the patients show no excitation, they tolerate the operation well without complaining of pain, and they are not restless after the operation. The term potentiated anesthesia seems

justified, because the drugs alone have little or no narcotic effect, but they increase the effect of anesthetic or narcotic drugs in a manner which corresponds to potentiation rather than simple addition.

Gyula Lugossy.

Cuperstein, R. I. **Affections of the eyes in the syndrome of temporal arteritis.** Vestnik oftal. **2**:11-17, May-June, 1958.

Four patients, aged 66 to 83 years, with ocular findings in association with temporal arteritis are described. The extraocular findings and symptoms consisted of headache, subfebrile temperature, hypochromic anemia, shift of leukocytic formula to the left, dilatation and tortuosity of the temporal arteries, the histopathologic finding of occlusion and recanalization of the lumen of the temporal artery and infiltration of its walls with giant and other inflammatory cells. The etiology was not established. The clinical course and histopathologic findings suggested infections or allergic-infectious etiology.

Blindness or marked reduction of vision occurred in all patients. In two there was complete loss of vision in both eyes, in one complete blindness in one eye and marked visual loss in the other, and in one there was marked visual loss in both eyes. Ophthalmoscopic findings in one patient who died consisted of attenuations of the retinal vessels. In two patients there was swelling of the disc with secondary atrophy of the nerve. In one patient swelling of the disc and retina was followed by optic atrophy. Since treatment could not be based on recognition of the cause the usual anti-infection therapy became necessary; anti-coagulants and cortisone preparations were also used and the temporal artery was resected. (6 figures, 28 references).

Victor Goodside.

Dekking, H. M. **A self-compensating electronic manometer for the anterior chamber.** Ophthalmologica **135**:251-255, April, 1958.

Dekking describes a manometer for the anterior chamber with automatic compensation. (3 figures)

F. H. Haassler.

De Rosa, L. and Sbordone, G. **Affections of the eye of focal origin.** Arch. di ottal. **62**:287-290, July-Aug., 1958.

In 50 cases of iritis or keratitis removal of foci of infection appeared to be indicated. The focus found was usually in the tonsils. (1 table, 4 references)

Paul W. Miles.

Flieringa, H. J. **Shortening of the eyeball.** Ophthalmologica **135**:255-260, April, 1958.

Flieringa describes a new operation for shortening the eyeball by bringing about permanent scleral folds. The procedure is made clear with the help of diagrammatic drawings. (11 figures)

F. H. Haessler.

Grósz, I., Turi, K. and Dózsán, G. **Evaluation of antistreptolysin reactions in diseases of the eyes.** Szemeszet **95**:137-143, Dec., 1958.

The antistreptolysin reaction was evaluated in the serum of 40 patients; 22 patients had iritis, 14 iridocyclitis, and 4 phlyctenular keratoconjunctivitis. Their ages ranged from 14 to 65 years. The reaction was positive in seven cases of iritis, five of iridocyclitis, and three of phlyctenular disease. In order to follow the titer dynamically, two or more examinations were made in each case. The authors conclude that this reaction, like all laboratory examinations, is to be evaluated with the whole pattern; alone it has little value. Usually it is positive when there is an active streptococcic focus; a negative result also is a valuable help. The sedimentation rate has not become superfluous.

Gyula Lugossy.

Konstas, K. A. **Fundus changes in pulmonary tuberculosis.** Ophthalmologica **135**:187-192, March, 1958.

Abnormalities of the fundus were found in 43 of 318 patients with pulmonary tuberculosis, in nearly 13 percent. Most of the lesions, of which six were in the retina and 37 in the choroid, were clinically harmless. Among 100 healthy children five had similar choroidal lesions and three of the children with lesions had had active tuberculosis in a lung or lymph node and the two others reacted positively to the Mantoux test. (3 references)

F. H. Haessler.

Lemoine, A. N., Jr., Robison, J. T., Jr. and Calkins, L. L. **A scleral imbrication technique.** A.M.A. Arch. Ophth. **60**:237-238, Aug., 1958.

The authors describe a method of scleral resection and incarceration of a roll of sclera. (1 figure) G. S. Tyner.

Malanove, N. L. **Use of nicotinic acid in streptomycin therapy of tuberculous chorioretinitis.** Vestnik oftal. **2**:39-41, Sept.-Oct., 1958.

Simultaneous administration of nicotinic acid and streptomycin in experiments on animals was found to produce an increase in streptomycin in the ocular aqueous and blood serum. Accordingly nicotinic acid was administered together with streptomycin in the treatment of tuberculous chorioretinopathy in 70 patients. It was found that the course of the disease was shortened to three or four weeks whereas in a control group with streptomycin alone the active course lasted seven to eight weeks. The immediate visual result and the remote outcome were superior in the patients receiving nicotinic acid plus streptomycin.

Victor Goodside.

Marke, P. **Ultrasound in ophthalmology.** Szemlszet **95**:104-108, Sept., 1958.

The author summarizes the biologic effects of ultrasound, the methods of application used in ophthalmology, and the results obtained. He emphasises that the intensity of radiation must not exceed 1 w/cm². It has been applied to vitreous opacities, chalazeon, scars in the skin of lids, and the crystalline lens after the operation for congenital cataract. More recently it is used in localization of foreign bodies, measuring the axis of eyes, and diagnosis of retinal tumors and ablation. Malignant tumor is a serious contraindication to ultrasound irradiation; it has no selective destructive effect on tumor tissue and, on the contrary, proliferation of the tumor may result from the irradiation. Caution is imperative in hemorrhagic tendencies and in excessive myopia because of the danger of retinal detachment.

Gyula Lugossy.

Mis, Marian. **A simple instrument for objective marking of adaptation utilizing optokinetic nystagmus.** Klinika Oczna **28**: 95-100, 1958.

A simply-constructed adaptometer based on the observation of optokinetic nystagmus is presented. Nystagmus is observed by looking with an ophthalmoscope into the other eye. The instrument is described in detail. The principle is the same as that of the original adaptometer of Ohm. Instead of the rotating drum the author uses movable mirror. (6 figures, 9 references) Sylvan Brandon.

Mis, Marian. **Micrometer attachment for measurement of eyegrounds.** Klinika Oczna **28**:81-87, 1958.

An attachment to the head of the "Oculus" ophthalmoscope is described. Micrometric arrangement permits exact calculation of the size of details in the fundus. Comparison and relative size of retinal changes can be measured by noting position of the scale immediately during the examination. (4 figures, 1 table, 10 references) Sylvan Brandon.

Mis, Marian. **Simple adaptometer with a scale for marking the adaptation curve.** Klinika Oczna **28**:87-94, 1958.

The author presents a self-registering adaptometer of a simple construction which is much cheaper than the Goldman adaptometer. (10 figures)

Sylvan Brandon.

Shibata, H. and Amano, K. **Objective measurement of ocular axis by X-ray photograph.** Jap. J. Ophth. **2**:263-268, Oct.-Dec., 1958.

The authors describe their method of measuring the axial length of the living eyeball objectively by X-ray photography. Air is injected under Tenon's capsule, a contact lens is placed upon the cornea, and the photograph is taken. The optical length is found by subtracting three percent from the apparent axial length plus 0.5 mm. for the thickness of the tin-impregnated corneal lens and one mm. for the thickness of the sclera and choroid. (4 figures, 1 table, 4 references)

Irwin E. Gaynon.

Smith, J. Lawton. **Pseudotumor cerebri.** Tr. Am. Acad. Ophth. **62**:432-440, May-June, 1958.

This condition is marked by signs and symptoms of increased intracranial pressure (papilledema, blurred vision, headache, nausea, vomiting, and visual obscurations); lack of focal neurologic signs (with the exception of abducens palsies); clear cerebrospinal fluid; and normal or small ventricles. Certain secondary cases, such as those due to aseptic venous sinus thrombosis, to otitic and sinus disease, and to premenstrual retention of fluid, may be excluded from the idiopathic group. Smith studied 36 cases, two of which were associated with persistent comitant esotropia. The latter two are presented in detail. The condition often occurred among obese, young, white females complaining of headache, blurred vision and diplopia. Papilledema averaged three diopters, and visual acuity was usually good. Despite studies of blood chemistry, nothing new in the etiology of the idiopathic cases has been found. (1 table, 27 references)

Harry Horwich.

Sugahara, A. and Hamada, M. **Sensitivitor of the field of vision.** Acta Soc. Ophth. Japan **62**:1902-1909, Oct., 1958.

A new test object for visual field examination on a campimeter is described. The test object is illuminated from a built-in lamp. The intensity of the illumination can gradually be changed from 0 to 11.5 lux. It is designed to measure, at various points of the central visual field, the minimum illumination of the object which can be recognized. The test should be done in a room illuminated at 300 to 500 lux. By this method a decrease in the sensitivity of the retina can sharply be detected, and it is an aid in early diagnosis of some conditions. Some examples are given. In the early stage of glaucoma a definite decrease in the sensitivity can be demonstrated in the peripapillary retina before enlargement of the blind spot. A hemianoptic decrease in sensitivity is demonstrated in some cases of brain diseases considerably earlier than the appearance of actual hemianopsia. (5 figures, 2 tables, 8 references)

Yukihiko Mitsui.

Sundmark, Eric **Recording of the human electroretinogram with the contact glass. II. Potential drops between retina and electrodes.** Acta ophth. **36**:829-836, 1958.

The ERG was recorded before enucleation from a human eye with a melanoma of the choroid, both in the standardized clinical manner and with special electrodes applied externally at the posterior pole and in the vitreous. About one third of the potential difference after light stimulus, measured between posterior pole and vitreous, is lost in the form of a potential drop when standard electrode

positions are used. (2 figures, 1 table, 5 references) John J. Stern.

Suzuki, I. and Satomura, S. **A study of the pulsation wave of the eye with ultrasonic interference method.** Acta Soc. Ophth. Japan **62**:1698-1701, Sept., 1958.

A new device to record pulsation of retinal vessels employing an ultrasonic interference method is described. The interference between the projected wave and the reflected wave from the retina is recorded. The authors consider that their method is best for recording the retinal pulsation wave. The curves obtained by their method are more accurate than those by manometry. The comparative data are illustrated. The method does not require surgical application. The ultrasonic wave required for this procedure is harmless. The pulsation wave of the retinal, the radial and the superficial temporal artery as recorded by the authors' device are compared with the ECG. The interval between the Q-wave of the ECG and the beginning of the pulsation wave of the artery is always constant for each artery. (5 figures, 8 references)

Yukihiko Mitsui.

Tanoue, T. **A study on the diagnosis of orbital tumors.** Acta Soc. Ophth. Japan **62**:1780-1802, Oct., 1958.

The potential difference between the frontal skin and that of the lid is measured. In normal persons the lid potential is negative. In cases of inflammatory conditions of the lids such as stye and lid abscess the negative lid potential increases. In lid and orbital tumors such as cancer, the negative potential decreases and often becomes positive. The measurement of lid potential may be an aid in the diagnosis of ocular tumors. (26 figures, 13 tables, 31 references)

Yukihiko Mitsui.

Uihlein, A. and Rucker, C. W. **The neurosurgeon's role in acute visual failure.** A.M.A. Arch. Ophth. **60**:223-229, Aug., 1958.

The conditions which may produce sudden decreased visual acuity are, in the order of their frequency: 1. pituitary tumor; 2. tumor of the optic nerve or chiasm; 3. supraclinoid aneurysm; 4. parasellar lesion; 5. thrombosis of the carotid artery; 6. hydrocephalus of the third ventricle; 7. chiasmal arachnoiditis; 8. fracture of the anterior cranial fossa; 9. basofrontal tumor of the skull; and 10. pseudotumor cerebri.

A diagnosis of retrobulbar neuritis or multiple sclerosis is commonly incorrectly made and may result in irreversible damage if the proper diagnosis is not made promptly. (8 figures and 5 references)

G. S. Tyner.

Yonebayashi, M. **Water content of the blood and the water-drinking test.** Acta Soc. Ophth. Japan **62**:1454-1463, Sept., 1958.

The author reports the result of a water-drinking test in 23 normal and five glaucomatous subjects. After the test the ocular tension increases in most glaucomatous eyes. The water content of the blood decreases for a certain period of time and then begins to increase. The ocular tension and the water content of blood do not increase in parallel. The same tendency is also seen in many of the normal controls. Yonebayashi believes that the increase in ocular tension after the water-drinking test is not necessarily due to hemodilution, but is partially due to a neurogenic influence. (5 figures, 3 tables, 46 references) Yukihiko Mitsui.

6

OCULAR MOTILITY

Borishpoletz, V. I. **Remote results of surgical treatment of concomitant strabismus.** Vestnik oftal. **2**:3-11, Nov.-Dec., 1958.

During the years 1948 to 1952, 938 pa-

tients were operated upon for concomitant strabismus. Of the 671 who had convergent strabismus, 455 obtained postoperatively a full cosmetic correction immediately, 176 showed under-correction and 40 showed over-correction. Of the 262 who had divergent strabismus, 192 obtained a good cosmetic result, 57 were under-corrected and 18 were over-corrected. The author was able to follow 248 of these patients for a determination of the outcome four to eight years after surgery. They had not in the interval received orthoptic training or any treatment other than refraction. The remote results showed disturbing differences from the immediate postoperative condition. Of the 123 patients who had had a good cosmetic result after surgery for convergent strabismus only 86 continued to have it. Of the 41 patients who had good cosmetic correction of their divergent strabismus, only 19 retained it years later. The type, number of operations, degree of squint or age of the patient had no significant effect on the eventual result. The acuity of vision of the deviating eye however did play a significant role in that the best cosmetic results were obtained where the visual acuity of the deviating eye was .2 or more. (5 tables, 12 references)

Victor Goodside.

De Conciliis, U. **Clinical syndrome of congenital retraction of the eyeball.** Arch. di ottal. **62**:309-315, July-Aug., 1958.

The Turk-Duane syndrome has been attributed to birth trauma, aplasia of oculomotor nuclei, or to anomalies of the insertion or form of a muscle. Malbran described three types. In addition to the usual variety he found a rare case of divergence in primary gaze, and another of vertical muscle paralysis. The author presents still another variation. A 14-year-old girl showed divergent strabismus in primary gaze and had no adduction or convergence. When she tried to abduct there was retraction of the globe and when she tried to adduct the globe slowly rotated vertically. Electromyography showed no innervation of the left lateral, left medial, and right medial rectus muscles. (2 figures, 40 references)

Paul W. Miles.

Ehrich, Wulf. **The influence of a strabismus operation on the fixation of the amblyopic eye.** Klin. Monatsbl. f. Augenh. **133**:846-848, 1958.

The operation alone improved the fixation in 40 out of 74 patients. In only five patients was the type of fixation poorer after the operation than before. Excentric fixators had a better chance of improvement in this series than central fixators. None of the children had any orthoptic or pleoptic treatment before the operation. (2 tables, 9 references)

Frederick C. Blodi.

Jaensch, P. A. **Remarks on the Hertwig-Magendie syndrome.** Klin. Monatsbl. f. Augenh. **133**:866-869, 1958.

Skew deviation is thought to be extremely rare and the recently described cases by Piper (Klin. Monatsbl. f. Augenh. 132:671) and by Jaensch do not belong to this syndrome. (21 references)

Frederick C. Blodi.

Katō, K. **The binocular movement of squint.** Acta Soc. Ophth. Japan **62**:2001-2029, Oct., 1958.

Katō introduces a new method for the determination of concomitant movement of the eyes. Results of measurement in 108 squint cases are described. Most cases of "concomitant squint" are not actually concomitant. Only 10 percent of the usual strabismus is actually concomitant. The Hirschberg method is not ideal for the determination of concomitant movement of the eyes; 78 percent of the concomitant cases as determined by Hirschberg's method are not actually concomitant

when measured by the author's method. (90 figures, 3 tables, 21 references)

Yukihiko Mitsui.

Matteucci, Pelegrino. **The unknown and the promise in the problem of strabismus and amblyopia.** Rassegna ital. d'ottal. **27**: 241-254, July-Aug., 1958.

The author gives a comprehensive review of the various theories of the etiology of the amblyopia of strabismus. He urges that intensive studies be made of the child before the age of two and a half years. The prophylaxis against amblyopia must be pointed fundamentally to the earliest correction of the refractive error, the anisometropia, heterophoria and finally to the amblyopia itself.

E. M. Blake.

Spanyol, V. **Enophthalmos and disappearance of orbital fat after surgery for strabismus.** Ophthalmologica **135**:223-226, March, 1958.

The author describes this rare occurrence which developed in the course of several years after surgery. (1 figure)

F. H. Haessler.

Vila Coro. **Supranuclear disturbances which affect ocular motility.** Arch. Soc. oftal. hispano-am. **18**:968-982, Sept., 1958.

The neuro-anatomy related to neuro-ophthalmology and to disturbances of ocular motility is described in detail.

Ray K. Daily.

7

CONJUNCTIVA, CORNEA, SCLERA

Belmonte, Jose. **Gastric juice injury of the cornea.** Arch. Soc. oftal. hispano-am. **18**:950-951, Sept., 1958.

Inadvertently some gastric juice had got into the left eye of a patient when he vomited during the induction of anesthesia for a gastrectomy which lasted over an hour. When seen three days later the left cornea had extensive erosion, with extensive loss of epithelium and stroma, marked conjunctival hyperemia, and folds in Descemet's membrane. It took a month for the lesion to heal. The author attributes the injury not to the hydrochloric acid alone, but to a true digestion of the corneal tissue by the gastric juice. (2 figures)

Ray K. Daily.

Bosso, Giancarlo. **Episcleritis in the course of polymorphic erythema.** Rassegna ital. d'ottal. **27**:292-303, July-Aug., 1958.

A 46-year-old woman had an acute gastro-intestinal disturbance with patches of intense redness on the hands and feet which were followed by arthritic changes. Soon after triangular patches of redness of the bulbar conjunctiva between the upper and lower lid margins were noted. A histologic examination showed the conjunctival epithelium intact but beneath this membrane there was inflammatory dissolution of the episcleral lamella, with foci of historeticular elements. There was one episcleral nodule. The author is unable to explain the restriction of the inflammatory changes in the bulbar conjunctiva to the exposed portion of the globe. (6 figures, 14 references)

E. M. Blake.

Carlberg, O. **Oculoglandular tularemia.** Acta ophth. **36**:815-818, 1958.

In a rare case of conjunctival participation in serologically proved tularemia, a pseudomembranaceous conjunctivitis with swelling of the preauricular glands was followed by conjunctival ulceration. The administration of streptomycin, penicillin and sulfonomide drops was followed by recovery. (2 figures, 16 references)

John J. Stern.

D'Arrigo, P. **The PCR in vernal conjunctivitis.** Arch. di ottal. **62**:291-300, July-Aug., 1958.

PCR, the protein-C-reaction, is a phenomenon of precipitation of a substance obtained from pneumococci by the serum of a patient subject to some active inflammation or serious disease such as a malignancy or a cardiac infarct. In a series of 22 patients with vernal conjunctivitis, the PCR was compared to serum protein determinations by paper chromatography and by other methods. The positive PCR findings indicated that vernal conjunctivitis is accompanied by systemic changes, possibly of the reticulo-endothelial system. (1 table, 38 references)

Paul W. Miles.

Dohlman, C.-H. and Larsson, S. **Megalocornea and cataract.** Acta ophth. **36**: 845-848, 1958.

A pedigree of four generations with eight cases of megalocornea is presented. In one patient cataracts were successfully removed in both eyes. Flieringa's ring is recommended in such cases to prevent loss of vitreous. (2 figures, 9 references)

John J. Stern.

Frasca, Gennaro. **Marginal degeneration of the cornea considered as a collagen disease.** Rassegna ital. d'ottal. **27**:245-273, July-Aug., 1958.

The author describes a case of marginal degeneration of the cornea in a man with primary chronic polyarthritis. Basing his consideration upon clinical and histopathologic evidence he concludes that the marginal degeneration of the cornea must be considered as an expression of fibrinoid necrosis of the corneal stroma with consequent cellular proliferation of a granulomatous type and sclerosis of the lamella. The illustrations show the gross and the microscopic changes. (8 figures, 36 references)

E. M. Blake.

de'Gennaro, Giuseppe. **Lysozymes in the treatment of corneal lesions due to chemical and physical agents.** Rassegna ital. d'ottal. **27**:274-291, July-Aug., 1958.

Lysozyme is a bacteriostatic and antibiotic enzyme derived principally from amino acids and shows a particular efficacy in maintaining the health of the conjunctiva and cornea. It is active against the more usual pathogenic organisms, herpes virus, and chemicals. The effect of various chemicals such as acetone, hydrochloric and nitric acids, ammonia, calcium hydroxide and sodium hydroxide was studied with and without the use of lysozyme in rabbit eyes. One eye was treated with lysozyme and the other untreated. Those treated with the enzyme were greatly improved and recovered good function. (10 figures, 11 references)

E. M. Blake.

Hirotsuji, I. **Therapy of serpent ulcer caused by pseudomonas.** Acta Soc. Ophth. Japan **62**:2235-2240, Nov., 1958.

This is a study of experimental keratitis in rabbits by inoculation of heat-killed bacteria. Pseudomonas, staphylococcus and pneumococcus were used. An intra-corneal inoculation with heat-killed pseudomonas results in the onset of severe hypopyon keratitis. Staphylococcus causes merely a slight keratitis and pneumococcus causes no keratitis. Hirotsuji considers that antibiotic treatment shows a smaller clinical effect in pseudomonas infection of the cornea, even if the organism is susceptible to the antibiotic used. (11 figures)

Yukihiko Mitsui.

Irinoda, K. and Mikami, H. **Angular blepharoconjunctivitis and pyridoxine (vitamin B_6) deficiency.** A.M.A. Arch. Ophth. **60**:303-311, Aug. 1958.

The authors conclude from their experiments that blepharoconjunctivitis angularis is caused chiefly by pyridoxine deficiency and that H. duplex infection has only a secondary significance in this disease. (4 figures, 7 tables and 21 references)

G. S. Tyner.

Kahan, A. and Beladi, I. **Clinical and etiologic evaluation of an epidemic of keratoconjunctivitis.** Ophthalmologica **135**: 79-87, Feb., 1958.

The authors report their study of 234 patients in an epidemic of keratoconjunctivitis during the years 1954 to 1956. Early in the epidemic foci in the cornea and limbus and eruption of the skin of the lids predominated. After April, 1956, the disease was characterized by numerous petechiae near the lid margin, conjunctivitis, pharyngitis, and such general symptoms as fever and somnolence. The patients were predominantly young people but the cornea was affected only in adults or in children who had a herpes eruption simultaneously. (3 figures, 2 tables, 15 references) F. H. Haessler.

Kamata, W. **A successful case of heterokeratoplasty.** Acta Soc. Ophth. Japan **62**: 2241-2244, Nov., 1958.

A man, 54 years of age, had a superficial leucoma of the cornea. A lamellar keratoplasty was made, using chicken cornea. The graft of chicken cornea is dipped into the patient's serum for 36 hours, in the refrigerator, before transplantation. The result is excellent. The vision improved from 0.04 to 0.2. The condition remained unchanged for 20 months. (3 figures, 10 references) Yukihiko Mitsui.

Kurus, Ernst. **Hyaline deposits in the cornea and the anterior chamber.** Klin. Monatsbl. f. Augenh. **133**:860-866, 1958.

Hyaline deposits in the corneal epithelium are found in absolute glaucoma. Larger deposits may occur in the stroma and on Descemet's membrane. Hyaline may occur free in the anterior chamber and one such patient is described. The patient had had a perforating injury 18 years earlier. The eye became inflamed and painful. An iridectomy was done and the grossly visible foreign bodies were washed out of the anterior chamber. They proved to be hyaline globules. (5 figures, 7 references) Frederick C. Blodi.

Leibiger, W. **Pigment deposits on the posterior corneal surface.** Klin. Monatsbl. f. Augenh. **133**:895-897, 1958.

Such deposits could be observed in 10 patients after a cataract extraction; in five of them the pigment was in or near the scar. Only one patient had diabetes. (1 figure, 7 references) Frederick C. Blodi.

Leiva, Modesto. **Multiple dermomas of the conjunctiva.** Arch. Soc. oftal. hispanoam. **18**:925-933, Sept., 1958.

The literature on the etiology of this neoplasm is reviewed, and three cases reported. One case was that of a 20-month-old child, with numerous congenital tumorous formations, in the right cornea, and the left upper lid, cornea and conjunctiva; there were neoplasms about the ears and scalp. The second case was that of a-nine-year old child with a large coloboma of the upper lid and a large dermoma on the infraexternal portion of the cornea, extending over the sclera and inner angle of the lids. The third case, a girl, twelve years old, had a lipodermoma of the cornea and sclera associated with pigment disturbances in the fundus. The diagnosis, pathology, etiology, and surgical treatment are discussed. (3 figures, 9 references) Ray K. Daily.

Lemmingson, W. and Riethe, P. **Mesodermal dysgenesis of cornea and iris combined with oligodontia.** Klin. Monatsbl. f. Augenh. **133**:877-891, 1958.

Three cases are described. This anomaly comprises malformations of the cornea, the chamber angle and the mesodermal part of the iris. The pupil is usually slitlike. These three patients also had glaucoma, progressive iris atrophy, myopia, ectopia of the lens and a reduced

number of teeth. Two patients had a microcornea. (11 figures, 1 table, 24 references) Frederick C. Blodi.

Marchessi, Fernando. **Therapy of subconjunctival hemorrhage.** Arch. Soc. oftal. hispano-am. **18**:934-939, Sept., 1958.

The author advocates subconjunctival injections of sterile air for the rapid absorption of subconjunctival hemorrhage; as recommended for this purpose by Fronimopulos, the injections should be superficial and directly over the hemorrhage. (10 references) Ray K. Daily.

Marin-Amat, M. **Herpetic keratitis and the phenomenon of virus interference.** Arch. Soc. oftal. hispano-am. **18**:939-949, Sept., 1958.

The author refers to an especially resistant case of herpetic keratitis cured by vaccination with smallpox vaccine and reported in a previous communication; he now reports two more cases treated by vaccination. One is a severe case of dendritic keratitis with participation of the iris and ciliary body; after vaccination with the smallpox vaccine, which took on the arm, the eye made a spectacular recovery. In the second case, that of disciform keratitis following a cement burn, vaccination was of little benefit. The author discusses the different response of eyes to this form of therapy. For vaccination to be beneficial to the course of the ocular lesion it is necessary that the vaccination take on the arm, indicating that the patient is in a state of receptivity, and that antibodies are being produced. If the patient is immune to the smallpox virus, as a result of a previous vaccination, there will be none or but a slight local response to the revaccination with no formation of antibodies and no effect on the ocular lesion. The literature on the phenomenon of interference of viruses and its mechanism is reviewed. (1 figure) Ray K. Daily.

Muscolino, F. and D'Arrigo, P. **Behavior of ionic iron and protoporphyrin IX freed from erythrocytes in vernal conjunctivitis.** Arch. di ottal. **62**:321-329, July-Aug., 1958.

Iron studies on erythrocytes from 18 children with acute primary vernal conjunctivitis showed an average hyposideremia of 76.38 percent. Ionized iron was increased, 349.20 percent while protoporphyrin IX was down 71.09 percent. Possible causes are discussed. (1 table, 23 references) Paul W. Miles.

Rollin, J. L. **A case of posterior embryotoxon.** Klin. Monatsbl. f. Augenh. **133**:897-899, 1958.

In a 19-year-old man Schwalbe's ring was conspicuously visible in both eyes. (3 figures, 8 references) Frederick C. Blodi.

Thygeson, P. and Nataf, R. **Etiologic problems in trachoma.** Rev. Intern. du trachome **35**:83-145, 1958.

The authors give a complete review of the present knowledge of the subject as well as of the research being done.

The viral nature of the Halberstaedter-Prowazek elementary bodies as well as their association with trachoma, their presence in experimental trachoma and their absence in the normal conjunctiva justify the conclusion that it is the cause of it. There is evidence that the initial body constitutes an early phase of intracellular development of trachoma virus and that the elementary bodies are derived from the initial bodies by binary fission; upon the cytoplasm of a cell they swell and start to multiply. The claim of the existence of a submicroscopic form of the virus is still dubious; with the electron microscope it is extremely difficult to differentiate between virus particles and other cell granules.

The analogy with other conditions such as molluscum contagiosum and some

types of keratoconjunctivitis suggests the possibility of a soluble necrotizing toxin but will not be subject to proof until the virus has been cultivated in quantity. The culture of the virus in quantity and capable of inducing experimental trachoma in man or monkeys has not yet been accomplished, and the criteria proposed by the authors for this purpose have not been fulfilled. Extreme care must be taken to differentiate experimental trachoma in animals from spontaneous folliculosis; they must be checked by human inoculations. It has been reported that succeeding inoculation of the virus tends to produce a disease with progressively milder onset and course; this would suggest the development of antibodies but this could also be explained on the basis of mobilization and persistence of leucocytes.

The therapeutic action of the sulfonamides is most probably due to the interruption of certain enzyme systems which prevent further intracellular multiplication of the trachoma virus; they do not seem to have a direct virucidal effect. Antibiotics seem to act in the same way.

Major differences in the clinical picture of trachoma in various parts of the world have suggested that more than one type of the disease exists. However, until serologic studies are performed to test the possibility of strain differences, it can only be said that the age of onset, host resistance, and degree of secondary infection explain such variability. There is no racial immunity although variations in the severity are common. The effect of poverty, promiscuity and ignorance are well known; however, dietary deficiencies do not seem to increase the susceptibility; it has even been suggested that actually malnutrition decreases it. (16 figures, 1 table, 118 references) José A. Ferreira.

8

UVEA, SYMPATHETIC DISEASE, AQUEOUS

Frasca, G. and Sola, M. **Research on the protein C reaction of aqueous in some inflammations of the anterior segment of the eye.** Arch. di ottal. **62**:331-338, July-Aug., 1958.

PCR, the protein-C-reaction, is a precipitation phenomenon of a substance obtained from pneumococci by the serum of a patient. PCR does not pass the blood-aqueous barrier, except in ciliary disease or inflammation. However, the authors found in a series of 20 cases of uveitis that the serum PCR was occasionally negative while the aqueous PCR was positive. (1 table, 17 references) Paul W. Miles.

Makley, T. A., Jr., and King, G. L. **Multiple cysts of the iris and ciliary body simulating a malignant melanoma.** Tr. Am. Acad. Ophth. **62**:441-443, May-June, 1958.

A 33-year-old woman complaining of a foreign body sensation and blurred vision despite 20/20 acuity was found to have a slight bulge in her iris. On dilatation, a dark, smooth, oval mass which did not transilluminate, was seen between the iris and the lens. P^{32} studies were not diagnostic. The mass increased during the next two months and the eye was enucleated.

Microscopic study showed a cyst of the iris epithelium reaching from the root to the sphincter. There was also marked cystic degeneration of the peripheral portion of the retina.

It is suggested that gonioscopy might have been of diagnostic value by demonstrating other cysts between the ciliary processes. (3 figures, 4 references)

Harry Horwich.

Rones, B. and Zimmerman, L. E. **The prognosis of primary tumors of the iris treated by iridectomy.** A.M.A. Arch. Ophth. **60**:193-205, Aug., 1958.

The Registry of Ophthalmic Pathology reports a ratio of 15:1 for the tumors of the ciliary body and choroid to those of the iris. There is also a marked difference

in their characteristics. Well demarcated tumors do not need to be treated at all unless they are at the periphery or are growing. With rare exceptions these tumors do not affect the patient's life expectancy. (3 figures, 13 tables, 11 references) G. S. Tyner.

Tenenbaum, E. and Kornblueth, W. **Cultivation of adult human iris in vitro.** A.M.A. Arch. Ophth. **60**:312-318, Aug., 1958.

In order to throw some light on the known lack of regeneration of iris tissue after iridectomy, attempts were made to culture the iris. They were successful but the growth of all the elements of the tissue was slow. Tissue taken during surgery for cataract grew more readily than that for primary narrow-angle glaucoma. (13 figures, 5 references) G. S. Tyner.

Tost, Manfred. **Rare tumors of the iris and the uvea, with pathologic examination. Klin.** Monatsbl. f. Augenh. **133**:848-860, 1958.

Three cases are reported. The first tumor occurred in a 50-year-old woman and proved to be a cyst of the pigment epithelium at the root of the iris. The second iris tumor was found in a 30-year-old man. It was either a neurinoma or a leiomyoma. The third eye was enucleated because a malignant melanoma was suspected. This eye had a congenital melanosis. A hemangioma and benign melanoma of the ciliary body were found. (13 figures, 58 references) Frederick C. Blodi.

Velhagen. K. **The treatment of choroidal detachment and postoperative intraocular hemorrhage.** Klin. Monatsbl. f. Augenh. **133**:776-784, 1958.

The author believes that choroidal detachment and expulsive hemorrhage are related conditions. In cases of hemorrhagic choroidal detachment a scleral trephine is indicated to evacuate old and new blood. Four examples are cited. The prophylactic scleral trephine is not advised. (1 figure, 38 references)

Frederick C. Blodi.

9

GLAUCOMA AND OCULAR TENSION

Colovine, S. **Wide antiglaucomatous posterior sclerotrepanation.** Arch. d'opht. **18**:185-189 March, 1959.

The author describes a trephine procedure by which a 4 mm. button is removed from the upper nasal sclera between the ciliary body and the ora serrata. The trephine opening is situated 6 or 7 mm. from the limbus between the superior and internal rectus muscles. The herniated retina and vitreous are touched with the actual cautery and the area is then covered with conjunctiva. The author reports in detail nine cases in which the results were favorable. Traumatic cyclitis occurs no more commonly in his procedure than in the classical trephine operation, and neither retinal detachment nor infection have occurred. (6 references)

P. Thygeson.

Junceda Avello, J. **Recent modifications in the calculations of ocular tonography.** Arch. Soc. oftal. hispano-am. **18**:336-342, April, 1958.

Junceda discusses the factors which mitigate against a correct interpretation of tonographic data. Attention is called to the modification in the tonometric tables in 1955, which must affect the tonographic tables. The tonometric tables calculated for standardized tonometers are invalidated by minimum variations in the construction of tonometers. The tables calculated for corneas 7.5 mm. in diameter are not applicable to microphthalmia or megalocornea. The manner in which tonography is performed must be taken into consideration and the tables should be corrected if tonography is performed with Goldman's applanation tonometer in the sitting position. The effect on the ocular

tension of variations in the general blood pressure should be compensated by eliminating the findings of the first minute of tonography, or beginning tonography half a minute after informing the patient that the examination is about to begin. The scleral rigidity and the venous episcleral pressure are sources of erroneous conclusions. The formulas for calculating the facility of outflow should be different for eyes with normal and abnormal ocular rigidity. (3 graphs) Ray K. Daily.

Lugossy, G. **Modified iridencleisis.** Szemeszet **95**:112-127, Sept., 1958.

By means of a modified iridencleisis the author succeeded in lowering ocular tension to normal in 40 eyes of 30 patients, of whom 12 were males and 18 females. The modification brings about the effect of iridencleisis and iridectomy. After preparing a large conjunctival flap, a scleral wound measuring 8 to 10 mm. is made with Landolt's lancet, 1.5 to 2 mm. above the limbus. In cases of a very narrow chamber, Gayet's incision is used. The iris is drawn out as in broad iridectomy and cut radially down to its root; one half is then pinched in the scleral wound with the pigmented epithelium layer facing the conjunctiva and finally laid upon the sclera. The conjunctival wound is united with interrupted or interrupted-continuous sutures. In 90 percent of the patients so operated upon the visual acuity and fields have not changed in several years, in 75 percent of them without miotic drugs. The modification, being a simple operation attended by no complications, may be used in all kinds of glaucoma.
Gyula Lugossy.

Pasino, Luigi. **Traumatic ocular hypertension with rubeosis iridis.** Rassegna ital. d'ottal. **27**:304-310, July-Aug., 1958.

In all cases of rubeosis iridis with increased ocular tension there is venous and capillary stasis, often requiring surgery for relief of the pressure and of the engorgement of the iris vessels. The case of a 46-year-old man is reported whose right eye was struck with a whip; pain and hypertension followed, but only after a week. Intense injection of the ciliary and iris vessels then developed; the pupil was oval and reacted slowly. The vitreous became cloudy but some retinal hemorrhages were observed. After a week of complete rest and the use of antibiotics and antihemorrhagic medication the intraocular pressure became normal and the vision 20/20. (2 figures, 15 references)
E. M. Blake.

Suda, K., Ikuta, M., Miyata, N., Furushima, M., Kihara, K. and Nakayama, M. **Prognosis of simple glaucoma.** Acta Soc. Ophth. Japan **62**:1491-1500, Sept., 1958.

The results in 80 eyes with simple glaucoma which have been observed for at least two years are summarized. In the early stage, a continuous application of miotics is enough to preserve the vision in most cases, but not the visual field. In 55 (80 percent) of 69 eyes which have been treated surgically a continuous normal tension was obtained. Iridencleisis and trephine operation give a better result than iridectomy and cyclodialysis. A continuous low tension, however, does not necessarily mean a preservation of vision. The vision was preserved in only 47 percent of the eyes operated upon. (24 tables, 18 references) Yukihiko Mitsui.

Yasuda, S. **The mechanism of reduction of tension by anthranilic acid.** Acta Soc. Ophth. Japan **62**:1475-1482, Sept., 1958.

This is a re-examination of Iinuma's claim that anthranilic acid is effective in the treatment of simple glaucoma. Yasuda demonstrates in rabbits that an administration of anthranilic acid causes a depression of aqueous formation and also a de-

crease in bicarbonate ion concentration in the aqueous, but does not cause an acceleration of aqueous outflow. It does not cause a depression of bicarbonate ion concentration in the blood. The author believes that the action of anthranilic acid is to cause a decrease of bicarbonate ions in the aqueous and thus to impede aqueous production. (6 tables, 59 references)

Yukihiko Mitsui.

10

CRYSTALLINE LENS

Agarwal, Lalit Prakash. **Cataract extraction after postplaced valvular iridencleisis.** Ophthalmologica **135**:91-94, Feb., 1958.

The author describes surgery for cataract in 106 patients who had had postplaced valvular iridencleisis. The operation for cataract did not interfere with the ocular tension or the adequacy of drainage which had been established by previous surgery. (2 figures, 1 table, 5 references) F. H. Haessler.

Barraquer, Joaquin. **Enzymatic zonulolysis. Contribution to the surgery of the crystalline lens (preliminary note).** Acta ophth. **36**:803-806, 1958.

A proteolytic enzyme, α-chymotrypsine, which is obtained from the pancreas of the calf, has been used to dissolve the zonular threads holding the lens in place. In rabbits the results were not conclusive, but the substance was shown to be innocuous to the intraocular structures. In enucleated human eyes, zonular lysis with spontaneous loosening of the lens was obtained. On blind human eyes, a 1:5000 solution injected into the pupil or behind the pupil loosened the lens sufficiently to allow easy intracapsular extraction with a suction cup. The author uses the method now as a routine in cataract extraction with uniformly good results. (11 references) John J. Stern.

Caballero del Castillo. **Ectoheterophakia.** Arch. Soc. oftal. hispano-am. **18**:962-967, Sept., 1958.

Three cases of ectopia lentis are reported, two in children with only the visual disturbances incident to this anomaly, and another with secondary glaucoma in the eye due to dislocation of the lens into the anterior chamber. Subsequently the lens of the second eye in this patient became spontaneously dislocated into the vitreous. The etiology, diagnosis, associated anomalies and treatment of this anomaly are discussed. Ray K. Daily.

Cogan, D. G., Stephen, J. F., Lubin, M., Donaldson, D. D. and Hardy, H. **Cataracts and ultra-high-frequency radiation.** A.M.A. Arch. Indust. Health **18**:299-302, Oct., 1958.

Rabbits were repeatedly exposed to ultra-high-frequency radiation (468 megacycles per second) in doses near the lethal level and distributed over the whole body. No cataracts developed after the exposures. (1 table, 7 references)

F. H. Haessler.

Del Castillo, Caballero. **Surgery of congenital cataract.** Arch. Soc. oftal. hispano-am. **18**:305-308, April, 1958.

The author's surgical pattern consists of a keratotomy with a keratome, insertion of a corneo-scleral suture, capsulectomy with capsule forceps, extraction of the lens masses with a suction tube held in the mouth, and irrigation of the anterior chamber. The advantages of this procedure over a discission are discussed.

Ray K. Daily.

Hernandez Guerra, F. and Jaime Hernandez, J. **Werner's syndrome in three brothers.** Arch. Soc. oftal. hispano-am. **18**:285-296, April, 1958.

This is a report of three cases of Werner's syndrome, with the objective of

placing them on record. The three brothers have two healthy brothers and one normal sister. One brother, 31 years old, had juvenile rapidly progressive bilateral cataracts, scleroderma, premature senility, poorly developed secondary sexual characteristics, ulcerated vocal cords, and diabetes. Discissions and irrigation of the anterior chamber was followed by a satisfactory visual acuity. Ten years later the visual acuity of the left eye failed because of a cystoid cicatrix and corneal infiltration. The fundus was senile with hardened arteries, loss of macular reflexes, and capillary degeneration. A brother, 28 years old, also lost vision because of cataracts; he had the same constitutional symptoms as the brother. His cataracts were dealt with by keratotomy, iridectomy, capsulotomy and extraction of the nucleus. Recovery was uneventful, and the visual acuity was satisfactory. The fundus exhibited marked senility. Another brother had the same history and was operated on by discissions. The etiology of this disturbance is briefly mentioned. (11 figures, 8 references) Ray K. Daily.

Schrader, Karlernst. **Diamox to prevent hemorrhage into the anterior chamber after cataract extraction.** Ophthalmologica **135**:182-186, March, 1958.

The use of diamox in the preparation of 106 patients for cataract surgery reduced the incidence of hemorrhage into the anterior chamber from almost 12 percent to 4.7. (5 references) F. H. Haessler.

Vancea, P. and Lazarescu, D. **A contribution to the study of lenticonus.** Ophthalmologica **135**:67-78, Feb., 1958.

The authors describe two cases of lenticonus, in one of which the changes affected both the anterior and posterior cortex. The embryonal nucleus was normal but there were cortical opacities and remains of the hyaloid artery. (4 figures, 59 references) F. H. Haessler.

Warmbt, W. and Nonnenmacher, H. **The influence of the weather on hemorrhages into the anterior chamber after cataract extractions.** Klin. Monatsbl. f. Augenh. **133**:821-835, 1958.

In 1957, 47 such hemorrhages occurred at the Dresden clinic among 278 cataract extractions. In 17 patients a definite exogenous reason for the bleeding could be found; the other 30 cases could be correlated with meterologic factors. Before or at the time of changing weather conditions, an increase of the hemorrhages was observed. (2 figures, 4 tables, 10 references) Frederick C. Blodi.

Wilson, W. A. and Donnell, G. N. **Cataracts in galactosemia.** A.M.A. Arch. Ophth. **60**:215-222, Aug., 1958.

Cataract was noted in eight of the 12 cases of galactosemia reported. No regression was noted in any patient, even under treatment. (3 figures, 17 references) G. S. Tyner.

NEWS ITEMS

EDITED BY DONALD J. LYLE, M.D.
411 Oak Street, Cincinnati 19, Ohio

News items should reach the editor by the 10th of the month. For adequate publicity, notice of postgraduate courses and meetings should be received three months in advance.

DEATHS

Dr. Ralph Allison Davis, Chicago, Illinois, died December 10, 1958, aged 59 years.

Dr. Thomas Holmes Emmens, Medford, Oregon, died November 8, 1958, aged 46 years.

Dr. Ralph Albert Fenton, Portland, Oregon, died November 2, 1958, aged 77 years.

Dr. Theodore Simon Kammerling, Chicago, Illinois, died November 20, 1958, aged 77 years.

Dr. Henry Charles Weber, Drexel Hill, Pennsylvania, died October 30, 1958, aged 71 years.

From *The Lancet* of January 31, 1959, it is learned that Mr. Philip Doyne, consulting ophthalmic surgeon to St. Thomas's Hospital and consulting surgeon to the Royal London Ophthalmic Hospital, died at his home at Henley-on-Thames on January 22nd, at the age of 72 years.

He was the son of Robert Doyne, founder of the Oxford Eye Hospital and the first master of the Oxford Ophthalmological Congress—an office which was later also held by Philip Doyne. From Winchester he went to Trinity College, Oxford, and to St. Thomas's Hospital, qualifying in 1913. Among the resident posts which he held was that of ophthalmic house-surgeon at St. Thomas's. His first hospital appointment was to the East London Children's Hospital; his interest in children's work continued, and he later became eye surgeon to The Hospital for Sick Children, Great Ormond Street.

During the 1914-1918 war he served with the R.A.M.C. in the Mesopotamian campaign. He ended his army service as an ophthalmic specialist, and he continued this work on his return to civil life. He was appointed assistant surgeon at Moorfields Eye Hosptial (as it was then called) in 1922 and ophthalmic surgeon at St. Thomas's two years later. Mr. Doyne leaves a widow and a daughter.

ANNOUNCEMENTS

COURSE IN LIGHT-COAGULATION

The fifth introductory course in light coagulation will be given at Bonn, Germany, from May 4 to 8, 1959.

On Monday, May 4th, the following subjects will be presented: "Technical details of the light coagulator" (there will be an opportunity to discuss various problems with Mr. Wolf, representative of the Zeiss Company); "Principles of light coagulation—demonstration of light coagulations and patients"; "Light coagulation in rabbits."

On Tuesday, May 5th, "Principles of light coagulation: a. Light coagulation for prevention and treatment of retinal detachment; b. Light coagulation combined with operative procedures"; "Demonstration of light coagulation and patients"; "Light coagulation in rabbits."

On Wednesday, May 6th, "Demonstration of in-patients"; "Light coagulation in Eales' diseases"; "Light coagulation in tumors"; "Demonstration of light coagulation and patients"; "Light coagulation in rabbits"; "Clinical conferences."

On Thursday, May 7th, "Light coagulation in tumors and other indications"; "Light coagulation of the iris"; "General discussion"; "Light coagulation of retina and iris in rabbits."

A boat trip on the Rhine has been scheduled for Friday, May 8th, as has a dinner in the "Lese-und Erholungsgesellschaft," Koblenzerstrasse, 35.

It has been requested that all who are interested in this course:

1. Write as soon as possible for hotel accommodations and for how many persons.
2. Bring your own ophthalmoscope; transformers for six and 12 volts are available.
3. There will be an excursion for the ladies to Cologne to visit the cathedral.
4. The fee of the course will be $50.00.
5. Those who wish to present a paper or to show a film on any interesting subject may do so at the clinical conference on Wednesday, May 6th, at 5:00 P.M. For reservations and information address: Prof. Dr. Meyer-Schwickerath, Poppelsdorfer Allee 106, Bonn, Germany.

CORNEO-PLASTIC CONFERENCE

Chairmen of sessions at the Corneo-Plastic Conference, Queen Victoria Hospital, East Grinstead, Sussex, June 26th and 27th will be: Dr. A. S. Parkes, London; Dr. R. Townley Paton, New York; Prof. E. B. Spaeth, Philadelphia; Prof. Derrick Vail, Chicago; Dr. J. Barraquer, Barcelona; Prof. Marc Amsler, Zurich.

The program will include: June 26th morning—Symposium on "Problems of tissue transplantation": "The biology of transplantation," Dr. L. Brent (University College); "The biological behavior of skin grafts," Mr. John Watson (East Grinstead); "Corneal transparency," Dr. Hugh Davson (University College); "The preservation of the living cell," Dr. Audrey Smith (Medical Research Council); *Discussion:* Openers, Mr. A. G. Leigh (London) and Mr. B. W. Rycroft (East Grinstead). June 26th afternoon—"Vitreous transplantation," Mr. P. McG. Moffatt (London); *Dis-*

cussion: Opener, Mr. C. Dee Shapland (London). There will be demonstration operations under closed-circuit television.

June 27th morning: Symposium on "Corneoplastic technique": "Some aspects of ptosis surgery," Mr. B. W. Rycroft (East Grinstead), "Problems of congenital defects of the lids and lid reconstruction," Sir Archibald McIndoe (East Grinstead), *Discussion:* Opener, Mr. H. B. Stallard (London); "Problems of lacrimal surgery," Mr. A. Werb (East Grinstead), *Discussion:* Opener, Prof. George Scott (Edinburgh); "Difficulties of socket reconstruction," Mr. G. J. Romanes (Roehampton), *Discussion:* Opener, Mr. Fenten Braithwaite (Newcastle).

June 27th afternoon: "Contact lenses and corneal grafts," Mr. F. Ridley (London). There will be a demonstration and discussion of individual cases in the lecture theater. During the conference, scientific exhibits from the Departments of Radiology, Pathology, and Photography will be on view. By courtesy of the Royal College of Surgeons of England (Department of Anatomy) illustrative dissections will also be available for inspection.

Glaucoma course at Massachusetts Eye and Ear Infirmary

A one-week course in glaucoma will be given at the Massachusetts Eye and Ear Infirmary under the direction of Dr. Paul A. Chandler, June 15 through June 20, 1959. Topics to be considered will be: Ocular hydrodynamics; gonioscopy, tonography, and perimetry; diagnosis, medical and surgical management. Surgical demonstrations will be held in the operating room. Patients will be available for demonstration and examination. Admission will be limited to 12 persons. Fee: $100.00.

Application may be made to: Dr. E. B. Dunphy, Chief of Ophthalmology, Massachusetts Eye and Ear Infirmary, 243 Charles Street, Boston 14, Massachusetts.

Home study courses

The 1959-1960 Home Study Courses in the basic sciences related to ophthalmology and otolaryngology, which are offered as a part of the educational program of the American Academy of Ophthalmology and Otolaryngology, will begin on September 1st and continue for a period of 10 months. Detailed information and application forms can be obtained from Dr. William L. Benedict, the executive secretary-treasurer of the academy, 15 Second Street, S.W., Rochester, Minnesota. Registrations should be completed before August 15th.

Wayne State University postgraduate course

The Department of Ophthalmology of Wayne State University College of Medicine will give a nine-month training in basic ophthalmology beginning September 21, 1959.

Six mornings a week are spent in lectures and laboratories. Each afternoon students are assigned to eye clinics of affiliated hospitals. A fundus clinic is held weekly; also, one on muscles.

Students are given 120 hours of training in physiologic optics, 122 hours in histology and pathology, 60 hours in biochemistry, 40 hours in neuroanatomy, and so forth.

Tuition is $300.00. Brochures on the basic sciences course will be mailed upon request. For further information write to: A. D. Ruedemann, M.D., chairman, Department of Ophthalmology, 690 Mullett Street, Detroit 26, Michigan.

Fellowship available

A one- to two-year hospital fellowship in ophthalmology, with special emphasis on plastic surgery, is available on July 1, 1959. Stipend. Also a two year-preceptorship is available. For further information write Dr. Wendell L. Hughes, 131 Fulton Avenue, Hempstead, New York.

Orthoptics course

The Basic Course in Orthoptics for Technicians, sponsored by The American Orthoptic Council, will be held in the Department of Ophthalmology, University Hospital, University of Michigan, from June 22 through August 15, 1959. As usual, there will be didactic lectures and practical demonstrations given by an outstanding faculty. Additional information as well as application blanks may be obtained by writing to Dr. John W. Henderson, University Hospital, Ann Arbor, Michigan.

Postgraduate courses

The following postgraduate courses will be offered by the Institute of Ophthalmology of the Americas: (1) Histopathology of the eye, May 18-23, 1959; (2) Review course covering practical aspects in perimetry, given evenings, May 20-22, 1959.

Further information regarding registration and fees may be obtained by writing to: Mrs. Tamar Weber, Registrar, Institute of Ophthalmology of the Americas, New York Eye and Ear Infirmary, 218 Second Avenue, New York 3, New York.

Clinical neuro-ophthalmology

A course in "Clinical neuro-ophthalmology" will be presented at the Massachusetts Eye and Ear Infirmary on May 17th through May 23rd (six days). The instruction will be provided by David G. Cogan, M.D., and associates.

The course will consist of a series of lectures and clinical demonstrations using patient material from the Massachusetts Eye and Ear Infirmary and the Massachusets General Hospital. Included in the course will be the essentials of ophthalmic neuroanatomy, methods of examination, and the diagnosis of clinical syndromes. Part of the instruction will be in the form of stereophotographs and motion pictures.

The tuition is $100.00 and the enrollment is limited to 30 members. Inquiries regarding the course may be addressed to: Charles Snyder, Registrar, Massachusetts Eye and Ear Infirmary, 243 Charles Street, Boston 14, Massachusetts.

Orthoptic examinations

The annual examination of orthoptic technicians by the American Orthoptic Council will be conducted in August and October, 1959.

The written examination will be nonassembled and will take place on Thursday, August 20th, in certain assigned cities, and will be proctored by designated ophthalmologists.

The oral and practical examinations will be on Saturday, October 10th, in Chicago just preceding the meeting of the American Academy of Ophthalmology and Otolaryngology.

Application for examination will be received by the office of the Chairman of Examinations, Frank D. Costenbader, M.D., 1605 22nd Street, N.W., Washington 8, D.C., and must be accompanied by the examination fee of $30.00. Applications will not be accepted after July 1, 1959.

Institute of Ophthalmology of the Americas

The Institute of Ophthalmology of the Americas announces that the second series of postgraduate courses for ophthalmologists will be given from September 14 to November 25, 1959.

Courses will be offered in the following subjects: "Advances in ocular prosthesis"; "Anomalies of extraocular muscles, including ptosis"; "Biomicroscopy"; "Biomicroscopy using near ultraviolet cobalt blue and polarized light"; "Clinical bacteriology"; "Complications of ophthalmologic surgery"; "Contact lenses"; "Electrophysiology and applied physiology of the eye"; "Enucleation and evisceration"; "Fundamental principles of orthoptics"; "Glaucoma"; "Gonioscopy and tonography"; "Keratectomies and keratoplasties"; "Lacrimal sac surgery"; "Low vision aids"; "Ocular biochemistry"; "Ocular geriatrics"; "Ocular microbiology"; "Ocular neuro-ophthalmology"; 'Ocular photography"; "Ocular radiology"; "Ocular therapeutics"; "Ophthalmoscopy"; "Pathology"; "Perimetry"; "Physiological optics"; "Plastic eye surgery"; "Pleoptics and macular function testing"; "Psychosomatic factors in ophthalmology"; "Radioisotopes in ophthalmology"; "Recent advances in cataract surgery"; "Refraction"; "Retinal detachment"; and "Surgery of the orbit."

Further information regarding the courses may be obtained by writing to: Mrs. Tamar Weber, Registrar, Institute of Ophthalmology of the Americas, New York Eye and Ear Infirmary, 218 Second Avenue, New York 3, New York.

Sight-saving conference

The latest developments and findings in the field of prevention of blindness were discussed at the 1959 annual Sight-Saving Conference of the National Society for the Prevention of Blindness, February 25th, 26th, and 27th at the Statler Hilton Hotel, New York. Ophthalmologists taking part in the meeting were Murray F. McCaslin, Pittsburgh; Arthur H. Downing, Des Moines; Samuel Lossef, New York; Ralph W. Ryan, Morgantown, West Virginia; Algernon B. Reese, New York; Graham Clark, New York; Donald A. Fonda, Ridgewood, New Jersey; Arnold S. Breakey, New York.

Pacific Coast meeting

The Pacific Coast Oto-Ophthalmological Society will hold its annual meeting May 3rd to 7th at the Hotel Riveria, Las Vegas Nevada. Guest Speakers include: R. R. Newell, M.D., professor of radiology and biophysics, Stanford, University School of Medicine; William P. Mikkelsen, M.D., associate clinical professor of surgery, University of Southern California; James F. Crow, Ph.D., professor and chairman, Department of Medical Genetics, University of Wisconsin; Stuart C. Cullen, M.D., professor of anesthesia, professor and chairman of Department of Anesthesia, University of California Medical Center; George E. Shambaugh, Jr., M.D., professor and chairman of Department of Otolaryngology, Northwestern University Medical School, Chicago; Sidney Riegelman, Ph.D., University of California School of Pharmacy; Lawrence R. Boies, M.D., professor and head of Department of Otolaryngology, University of Minnesota, Minneapolis; Dean M. Lierle, M.D., professor and head of Department of Otolaryngology and Maxillo-Facial Surgery, State University of Iowa, Iowa City; A. Edward Maumenee, M.D., professor and head of Department of Ophthalmology, Johns Hopkins University, Baltimore; John W. Henderson, M.D., Mayo Clinic, Rochester, Minnesota; Digby Leigh, M.D., chief of anesthesiology, Childrens Hospital, Los Angeles.

Irish Ophthalmological Society

The Irish Ophthalmological Society will hold its annual meeting in the Institute of Clinical Science, Belfast, on May 14th, 15th, and 16th. This meeting is being held in conjunction with a meeting of the Section of Ophthalmology, Royal Society of Medicine. Dr. Joaquin Barraquer, Barcelona, is to deliver the James Craig Lecture.

Miscellaneous

Glaucoma identification card

A glaucoma identification card for nationwide distribution was announced today by The National Medical Foundation for Eye Care as a major public service project. The card, similar in purpose to the diabetes identification card, will alert examining physicians that the patient has glaucoma and is using drugs. The names of the patient and of the ophthalmologist who prescribed the drugs, appear on the card, together with the prescription. This information helps to forestall the use of any contraindicated medication by the examining physician. Glaucoma patients who run out of their prescribed medicine while away from home are able to get a new supply quickly, without interrupting treatment, an important factor in glaucoma therapy.

In addition to the prescription for drugs, the glaucoma card also carries the spectacle prescription of the patient, which enables him to replace broken lenses when away from home.

The National Medical Foundation for Eye Care

glaucoma cards were printed as a public service by Abbott Laboratories, and are being initially distributed to physicians by the Laboratories. Packets of the cards may be obtained by writing to the Foundation office, 250 West 57th Street, New York 19, New York, or directly to Professional Services, Abbott Laboratories, North Chicago, Illinois.

Walter W. Wright Lectureship

Dr. Walter W. Wright, University of Toronto professor emeritus of ophthalmology, has been honored by a lectureship in his name established with funds raised by Department of Ophthalmology staff members, former students, and friends.

Dr. Wright conceived and developed the postgraduate training of physicians in eye diseases, the first such training in Canada, which began at the University of Toronto in 1941. Since then 65 specialists have been trained and are now practicing from coast to coast. This project still holds Dr. Wright's special interest. He is also consultant to the Canadian National Institute for the Blind, the Toronto General Hospital, and the Hospital for Sick Children.

He joined the University of Toronto staff in 1908 after being graduated four years earlier, and in 1914-1915 did postgraduate work at the Royal London Ophthalmic Hospital. He then served as an ophthalmic specialist in the Canadian Army Medical Corps at Bramshott Hospital, England, and in France, and later at Westcliff Hospital, Folkstone, Kent. He returned to the university in 1921, and headed the Department of Ophthalmology from 1941 until retirement in 1946.

Dr. Wright was the first to report on the use of living sutures in the treatment of ptosis in 1922, and in later years was active in stressing the necessity for early treatment of strabismus in infants. A renowned wicket-keeper in his younger days, Dr. Wright was a member of the Canadian cricket team versus the United States in 1905, and toured England in 1910.

The first Walter W. Wright Lecture was given by Prof. Norman Ashton, ophthalmic pathologist of the Institute of Ophthalmology, London, England, who spoke on "A new approach to the problem of diabetic retinopathy," at the Academy of Medicine on February 27th. In Britain Professor Ashton's research achievements have earned him the Nettleship Medal (1954), the Middlemore Prize (1955), and membership of the Royal College of Physicians of London. In 1957 he became the first non-American to receive the Proctor Medal of the American Association for Research in Ophthalmology.

Societies

Tunisian society

The Société Tunisienne d'Ophtalmologie will celebrate its third anniversary and the opening of the Ophthalmological Center with the first North African Seminar of Ophthalmology in Tunis on May 3rd through 6th. Algerian and Moroccan ophthalomologists will attend the meeting.

Pennsylvania meeting

The 1959 annual meeting of the Pennsylvania Academy of Ophthalmology and Otolaryngology will be held at the Bedford Springs Hotel, Bedford, Pennsylvania, on May 21st, 22nd, and 23rd. Ophthalmic speakers will include: Herbert J. Navyas, Philadelphia; J. Van Quereau, Reading; Allen W. Cowley, Harrisburg; Charles Jaeckle, East Orange; Joseph F. Novak, Pittsburgh; Robert Beitel, Jr., Allentown; Arthur Sherman, East Orange; Phillip Knapp, New York; William Krewson, III, Philadelphia; Edwin C. Tait, Norristown; Theodore Long, Lebanon; H. Walter Forster, Jr., Philadelphia; Joseph A. C. Wadsworth, New York; Joseph Alfano, Chicago; David Shoch, Chicago; Robert Davies, Pittsburgh. Moderators of the panel discussions will be: Paul Craig, Reading; Robert Shoemaker, Allentown; Samuel Phillips, Allentown; John Covey, Bellefonte; Isaac Tassman, Philadelphia; Joseph Morrison, Wilkes-Barre; Daniel S. DeStio, Pittsburgh; F. Johnson Putney, Wilkes-Barre; Raymond Jordan, Pittsburgh; B. L. Silverblatt, Pittsburgh.

Kansas City course

The University of Kansas School of Medicine in co-operation with the Kansas City Society of Ophthalmology and Otolaryngology and the Kansas Medical Society held a three-day course, April 8th through 10th. Guest instructors were Ralph W. Danielson, Denver; Fritz M. Jardon, Detroit; Irving H. Leopold, Philadelphia; A. Edward Maumenee, Baltimore; Robert N. Shaffer, San Francisco. Faculty from the University of Kansas Medical School included Maxwell G. Berry, Russell E. Bridwell, Larry L. Calkins, John I. Davies, Donald C. Greaves, A. N. Lemoine, Jr., Charles M. Poser, James T. Robison, Jr., and Dick H. Underwood.

Midwestern Research Section

Papers presented at the 11th annual meeting of the Midwestern Section of the Association for Research in Ophthalmology, Colorado University, Denver, April 18th and 19th, were:

"Autonomic conditioning in hysterical amblyopia," Robert A. Waggoner and T. F. Schlaegel, Jr., Indianapolis; "Retention and interocular transfer of intensity discrimination in dark-reared kittens after ablation of visual cortex," Louis Aarons and Austin H. Riesen, Chicago; "The role of increased responsibility in the precipitation of endogenous uveitis in adults," Evelyn Dunbar and T. F. Schlaegel, Jr., Indianapolis; "The electroencephalogram in retinitis pigmentosa," Alex Krill, Chicago; "The electromyography of vergence movement," James Miller, Saint Louis; "Extraocular muscle changes in thyro-pituitary disease," R. O. Schultz, M. W. Van Allen, and F. C. Blodi, Iowa City; "On the production of cataracts by the intralenticular injection of certain enzymes and enzyme inhibitors," David Shoch and E. Albert Zeller, Chicago; "Histology of zonulysis with chymotrypsin," Albert P. Ley, Ake Holmberg, and Tsuyoshi Yamashita, Saint Louis; "The effect of alpha-chymotrypsin (Quimo-

trase) on the rabbit eye," J. C. Thorson and P. J. Leinfelder, Iowa City:

"The fine structure of the ciliary epithelium," Ake Holmberg, Saint Louis; "Histochemical staining of ciliary body," Tsuyoshi Yamashita, Paul Cibis, and Bernard Becker, Saint Louis; "The effect of intraocular pressure on the resistance to outflow in the cat and rabbit," Mansour F. Armaly, Iowa City; "Studies on the effects of cholinergic organic phosphates on the eye: A preliminary report," Maurice Kadin and Kenneth DuBois, Chicago; "Parasympathetics and the facility of outflow: Effect and mechanism," Mansour F. Armaly, Iowa City; "The effect of carbonic anhydrase inhibitors on urinary citrate excretion by human, rats, and rabbits," Marguerite A. Constant and Bernard Becker, Saint Louis; "Mechanism of immediate parasympathetic effect of intraocular pressure," Mansour F. Armaly, Iowa City; "Demecarium bromide in the therapy of glaucoma," Andrew Gay, Allen A. Kolker, Tracy Gage, and Bernard Becker, Saint Louis; "The mechanism of the parasympatholytic mechanism of DME in the eye and its effect on intraocular pressure," Robert D. Whinery and Mansour F. Armaly, Iowa City; "A clinical study of the consistency of 1955 calibration for various tonometer weights," Mansour F. Armaly, Iowa City.

"Low temperature crystallography of bovine vitreous," John A. Buesseler, G. L. Rapatz, and Ronald L. Engerman, Madison, Wisconsin; "The vitreous humor as observed with the interference microscope." Arlene Longwell, Argonne National Laboratory, Lemont, Illinois; "Flat preparation of rat retina stained with PAS," Ronald L. Engerman, John A. Buesseler, and R. K. Meyer, Madison; "Drug actions on isolated intraocular muscles of cats," Julia T. Apter, Chicago; "Iridopathy and retinopathy produced by deoxycorticosterone acetate in the rat," Ronald L. Engerman, John A. Buesseler, and R. K. Meyer, Madison; "The distribution of C^{14}-labeled atropine in rabbit eyes," R. G. Janes, Iowa City; "Induction and inhibition of iridopathy and retinopathy in the rat," Ronald L. Engerman, John A. Buesseler, and R. K. Meyer, Madison; "The use of succinlcholine in ocular surgery," William M. Lewalen, Jr., and B. L. Hicks, Pueblo, Colorado.

Nassau meeting

Dr. Sidney Fox, New York, presented a paper on "The handling of entropion and ectropion" at a recent meeting of the Nassau (County, New York) Ophthalmological Society, and Dr. Earl Lewis showed a movie on "Alphachymotrypsin."

Philippines society

The Ophthalmological Society of the Philippines, the first ophthalmic society in the Philippines, has recently been organized to elevate the standard of ophthalmic practice in the Philippines and to promote scientific and cultural ties with ophthalmic societies of other nations. Officers of the newly organized society, which is recognized by and affiliated with the Philippine Medical Association, are: President, Dr. Felisa N. Fernando; vice-president, Dr. Herminio Velarde, Jr.; secretary-treasurer, Dr. Severino P. Lopez. On the Board of Directors are Dr. Sabino Santos and Dr. Ramon Batungbacal.

Personals

Dr. George Wald, professor of biology at Harvard University, received the 1959 Rumford Premium of the American Academy of Arts and Sciences, Bsoton, on March 11th. Dr. Wald received the $5,000.00 award for his studies on the biochemical basis of vision.

Dr. Bradley Ralph Straatsma, New York, has been appointed associate professor of surgery at the School of Medicine, University of California at Los Angeles. He will succeed Dr. S. Rodman Irvine as chairman of the Division of Ophthalmology. Dr. Irvine will remain as clinical professor.

Dr. Lalit P. Agarwal has been appointed professor and head of the Department of Ophthalmology, All-India Institute of Medical Sciences, Ansari Nagar, New Delhi.

Dr. Windsor S. Davies, Detroit, was invited by the Pan-American Association of Ophthalmology to lecture in South America. He accepted and has lectured in São Paulo, Montevideo, Buenos Aires, Santiago, Lima, and Bogotá.

Dr. Frederick Stocker, Durham, North Carolina, lectured in Bogotá and Caracas at the invitation of the Pan-American Association of Ophthalmology.

Dr. Louis Girard, of Baylor University, Texas, was invited by the medical college of Recife (Brazil) to give a course on extraocular muscles. After that, at the invitation of the Pan-American Association of Ophthalmology, Dr. Girard, together with vice-president Nicholson, came to São Paulo to lecture.

Derrick Vail, M.D., professor and head of the Department of Ophthalmology, Northwestern University Medical School, Chicago, Illinois, will give the 1959 W. F. MacArthur Lecture in Medicine, the University of Edinburgh, Scotland, at 5:00 P.M. on June 18th. His subject will be "The congenital cataract and its surgery."

SOUTH-EAST METROPOLITAN REGIONAL HOSPITAL BOARD CORNEO-PLASTIC CONFERENCE

Preliminary Notice

A conference will be held on June 26 and June 27, 1959, at the Queen Victoria Hospital, East Grinstead, Sussex. There will be a symposium on "Problems of tissue transplantation" and lectures on surgery of the lids, cornea, lacrimal apparatus and socket. Illustrative cases, operations on closed-circuit television, and scientific exhibits will be shown.

The conference will be strictly limited to 60 members, including places reserved for visitors from abroad and for nonconsultant surgeons.

An inaugural dinner will be given to members and their wives on June 26, 1959. Excursions have been arranged to Canterbury and Petworth for the ladies.

Registration fees. Consultants, £5 5s. Od. Nonconsultants, £3 3s. Od. For further information please apply to the Secretary, Corneo-Plastic Unit, Queen Victoria Hospital, East Grinstead, Sussex.

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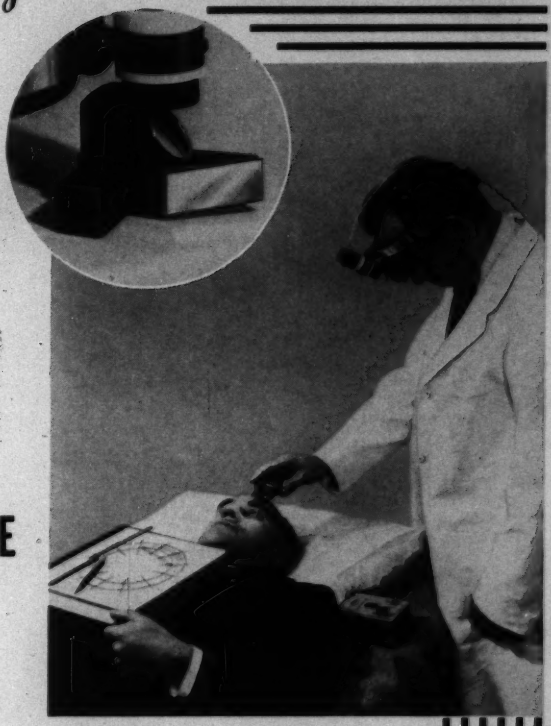

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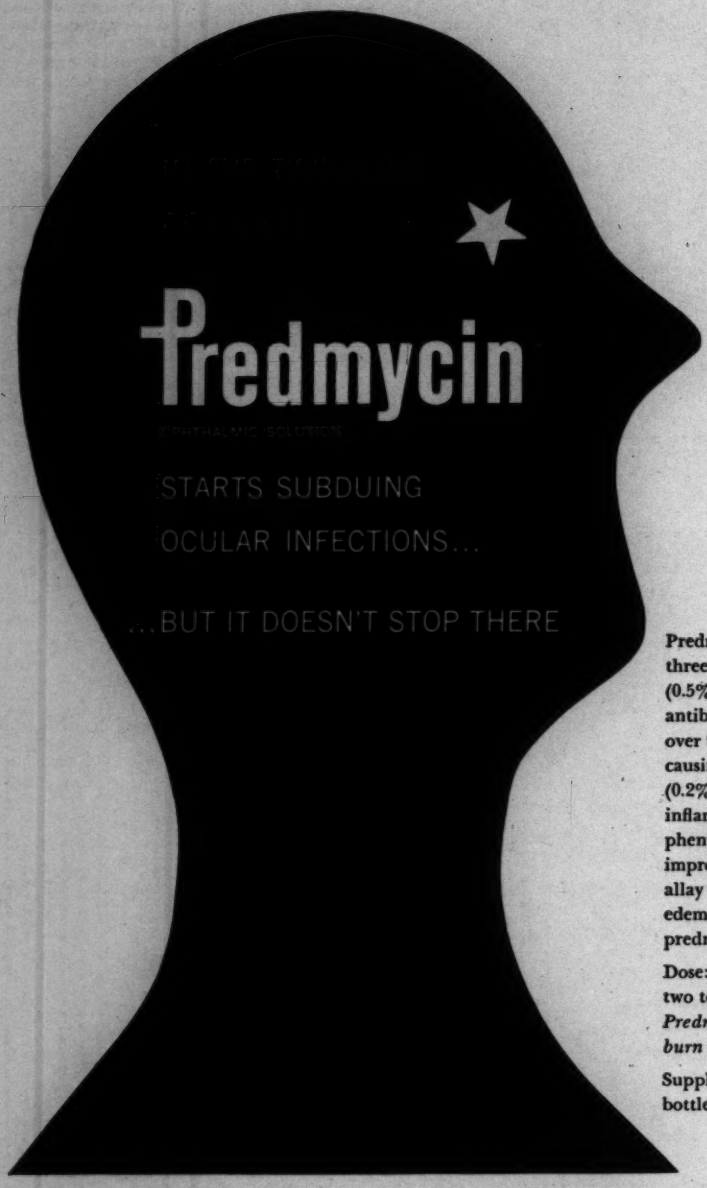

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